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ENCYCLOPÆDIA MEDICA

UNDER THE GENERAL EDITORSHIP OF
CHALMERS WATSON, M.B., M.R.C.P.E.

VOLUME V

HERPES TO *JAWS*

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Herpes.

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Herpes

Herpes is a term employed to designate an acute inflammatory affection, characterised by the development of vesicles closely grouped upon a patch of erythematous skin or mucous membrane.

Two forms of disease are recognised as coming under the above anatomical designation, but they differ widely as regards their clinical phenomena and pathological relations.

Herpes febrilis vel Catarrhalis

Febrile or catarrhal herpes frequently follows a rigor, and may be its sole sequela. Before the appearance of the eruption the temperature may be considerably raised, especially in children, but it usually falls on its appearance. Crops of herpes are of common occurrence in acute febrile diseases, accompanying their onset or crisis; they are most frequent in pneumonia, pleurisy, ague, enteric fever, and "catheter fever," but may accompany many indefinite gastro-intestinal disturbances. In other instances herpes may occur and recur frequently, often at definite intervals, without any rise of temperature or tangible determining cause. Many cases tend to occur in spring and autumn, and attain almost "epidemic" proportions. The lesions of febrile herpes do not show nerve distribution like those of zoster, from which they further differ in being frequently bilateral, sometimes even symmetrical, in being painless, leaving no scar, and in their great tendency to relapse.

Herpes facialis is the commonest form; it attacks in order of frequency the lips, the alæ nasi, the chin, and the ears; the "flush patches" in the malar region are also often involved. Patches may also appear on the tongue, soft palate, uvula, and tonsils generally in connection with digestive disorders, and as these soon give rise to minute ulcerations ("dyspeptic ulcers"), as the result of the maceration of their epidermis by the fluids of the mouth, they may be mistaken for aphthæ, diphtheria, or even syphilis. On the skin a slightly infiltrated reddish patch is at first present, accompanied generally by a little tension, burning, tingling, or itching. The patch is usually round or oval, and may be of very variable size; in a few hours minute vesicles form upon it, averaging about the size of a millet seed. Some of these vesicles usually coalesce, but at the margin they generally remain discrete. In two or three days the vesicles burst, their epidermis shrivels, their liquid contents dry up to form a thin scab; when this is removed a minute, shallow, polycyclical ulcer is exposed. At the end of a week or so the scab, which becomes daily thinner, finally separates, leaving only a pinkish or slightly pigmented spot which rapidly disappears. It is extremely rare, and in marked contrast to zoster, to have any permanent scar left even when the lesions have been the seat of pus infection, or been irritated by unsuitable dressings.

Herpes genitalis is a common disease in the male sex up to forty years of age, but in the female sex its incidence is almost entirely confined to prostitutes, occurring often at their menstrual periods, and frequently in those who have suffered from antecedent syphilis. In the male the disease usually occurs on the prepuce; it tends to relapse at much shorter intervals than herpes facialis, and is certainly of frequent occurrence in persons who have had syphilis. It sometimes recurs after every act of coitus in some individuals, irrespective of the health of the woman, and often follows indiscretions of diet, or may occur independently of any ascertainable cause. In France it is regarded as strong evidence of "arthritism" (*vide* "Gout," vol. iv. p. 213). The chief importance of progenital herpes lies in the facility for which it may be mistaken for soft chancres, especially when not seen at the commencement. Generally speaking, the lesion of chancroid is at first single, those of herpes are multiple, and on an erythematous base; the herpetic lesions are generally cleaner, not so purulent as the chancre; there is more burning and itching in herpes than in chancroid; finally, the pus of herpes is not auto-inoculable.

Treatment.—Facial herpes may often be abated in the early stage by mopping with swabs soaked in rectified spirit or eau de Cologne. Sometimes bathing with very hot water answers equally well, or the application of a coating of collodion.

In the later stages inert powders (oxide of zinc, starch, chalk, emol) may be dusted on; when the vesicles burst, dilute boric vaselin or ammoniated mercury ointment may be used. Norman Walker recommends painting the affected parts in each attack of facial herpes with argent. nitrat. (gr. xx.) in spir. æther. nitrosi (℥j.), as distinctly increasing the intervals between the attacks and ultimately sometimes effecting a cure. For the same purpose arsenic in prolonged courses and gradually increasing doses is warmly recommended in many quarters, while others rely upon nerve tonics (quinine, strychnine, belladonna, bromide of potassium) or change of air to various spas, home or foreign. In the management of genital herpes in both sexes strict attention to cleanliness is of paramount importance.

Herpes Zoster or Zona

Herpes zoster, zona, or shingles—so called from its girdle-like distribution—is a fairly common disease, and occurs in about 6 per cent of all skin cases (Radcliffe-Crocker).

Its etiology is obscure, and beyond cold or chill no definite cause can be assigned for it, although recent views tend to favour the possibility of its being caused by a specific poison. It occurs in both sexes, and is generally stated to be most frequent between the ages of 3 and 20, the maximum incidence being between the ages of 12 and 13; but older people are by no means exempt, and in them the symptoms are apt to be severe. Although the disease often occurs in persons in robust health, general debility acts as a predisposing cause, as also—as first pointed out by Hutchinson—does arsenic when taken over long periods of time; but that arsenic is only an indirect cause is clearly shown by the small number who are affected out of the multitude who take the drug.

Herpes zoster may be *symptomatic*, and, as such, is seen in certain diseases of the nervous system, as tabes dorsalis and myelitis; in the latter the eruption often corresponds to the upper level of the anæsthesia. Chronic peripheral irritation, *e.g.* caries of a rib or nerve injuries, will also sometimes give rise to attacks, which in such cases may be recurrent. The disease is probably of a specific nature, as shown by its acute onset, definite course, the infrequency of second attacks, and by the fact that it often recurs in distinct epidemics. Head considers it an acute inflammatory affection of the posterior root ganglia, analogous to that of the cells of the anterior cornua in acute anterior poliomyelitis.

Symptoms and Course.—An attack of herpes zoster is usually ushered in with general feelings of malaise and a rise of temperature, which, in children, may reach 102° F.; superadded to these symptoms being acute pain and hyperæsthesia over the nerve area subsequently to be affected. In children the amount of pain may be slight or nil. The date of the appearance of the eruption is variable, but it usually appears at earliest two or three days after the first symptoms have shown themselves.

The eruption appears first as an erythematous patch or patches, upon which numbers of small vesicles develop within a few hours. This outbreak follows the distribution of the nerve-fibres of the posterior roots, and is most intense where the main branches come to the surface, the patches being frequently oval, their long axis parallel to the subjacent nerve. The characteristic distribution is most typically shown in a case of intercostal herpes, in which one group of vesicles will often be seen near the spine over the spot where the posterior primary branch of the posterior root comes to the surface; another close to the axillary line over the site of the lateral branch of the anterior primary division; and a third group near the mid-line marking the termination of the anterior primary branch. There is no uniformity as to the order in which these maxima of intensity are affected, but as a rule the first patch to evolve is that nearest the nerve centre. The eruption is always most intense at these spots, and the intervals between them may or may not be filled up according to the severity of the attack. The right side is, curiously enough, much more commonly affected than the left. The corresponding lymphatic glands are almost always enlarged and tender, and may be so affected before the appearance of the eruption.

The eruption may continue to appear in crops extending over a week, or even longer, or it may be fully developed in two days; in the former case it appears early in the disease, and its evolution is accompanied by persistent pyrexia and malaise; in the latter it usually denotes the termination of the pyrexial period, and may be regarded as a "critical" phenomenon.

The initial lesions are erythematous patches, upon which papules and small vesicles rapidly form, which by coalescence may form comparatively large blebs. Some difficulty in diagnosis may occasionally be caused by the eruption being abortive, and appearing only as ill-developed grouped papulo-vesicles.

The vesicles are, at first, filled with clear fluid, which gradually becomes turbid, and dries up to form small scabs, which usually separate in about ten days. In more severe cases the vesicles, probably from extraneous causes, contain purulent or blood-stained fluid, often become gangrenous, and give rise to ulcers of various depths, which only heal with considerable scarring.

Herpes zoster may occur in the course of any cutaneous nerve, and is designated by various names according to the part affected, *e.g.* herpes ophthalmicus, cruralis, etc. It is especially frequent over the distribution of the intercostal, lumbar, and ophthalmic divisions of the fifth nerve, and is very rarely found below the knee. Herpes is comparatively frequent over the areas of distribution of the sciatic, anterior crural, ilio-inguinal, musculo-spiral, occipital, and superficial cervical nerves. Herpes of the ophthalmic division of the fifth nerve is especially important, as the subsequent scarring is generally deep and correspondingly disfiguring, and as the cornea is apt to be attacked, a complication which is most frequent in cases where the nasal branches of the nerve are affected. Zoster of the superior and inferior maxillary nerves is generally accompanied by lesions on the soft and hard palate, and on the tongue.

There are several very striking and important characteristics of attacks of zoster: the eruption is very seldom bilateral—though it may occasionally overlap the middle line—and it is never symmetrical; one attack almost invariably protects against a second, and in those cases in which second attacks do occur the eruption never occurs over the site of the former lesion.

Distribution.—Herpes zoster follows the distribution of the fibres of the posterior root ganglia, and not the course of peripheral nerves. This important fact was first pointed out by Baerensprung, and has recently been further extensively investigated by Head, who has used the distribution of the eruption as a means of mapping out the areas of skin supplied by the different posterior nerve roots.

Pathology.—Each vesicle consists of a small unilocular cavity containing fluid and altered epithelial cells with leucocytes; the papillæ of the skin form its floor. Several vesicles may coalesce to form multilocular blebs. Protozoa-like bodies described by Pfeiffer as pathogenic are probably only altered epithelial cells. The underlying papillæ are deeply congested and infiltrated with leucocytes. The peripheral cutaneous nerves show swelling of the neurilemma, degeneration of the medulla and moniliform swelling of the axis-cylinder. Ten days after the onset of eruption the larger branches show marked degenerative changes (Campbell and Head). Changes have now been definitely established occurring in the ganglia of the posterior nerve roots which extend for some distance up the posterior columns of the cord; the most common conditions found are hæmorrhages and inflammatory exudation, and secondary to these sclerosis, while degenerative changes have been traced from the posterior root ganglia through the posterior roots to the peripheral sensory nerves. In herpes ophthalmicus inflammatory changes are present in the Gasserian ganglion.

Complications and Sequelæ.—The amount of *scarring* varies considerably; in some cases there is scarcely any, and in others it is very deep, the preceding ulceration being sometimes very persistent.

Pain in the majority of cases disappears with the rash; in some, it persists for a considerable time after, and occasionally lasts for months or even years. This after-pain is a very troublesome symptom, which is of very common occurrence in elderly people, and in those cases where the disease has been followed by deep scarring.

The *ocular complications* of herpes of the ophthalmic division of the fifth are not infrequently serious, the most common being ulceration of the conjunctiva and cornea; iritis and panophthalmitis occasionally occur, and in very rare instances paralysis of orbital or ocular muscles have been met with.

Other motor paralyses are occasionally observed. Thus paralysis of the hand has been recorded after a herpes involving the cervical and post-dorsal nerves; facial paralysis of Bell's type has been stated to follow cervical herpes. On the other hand, Head states that "with the exception of the iris, there is no evidence that any deep structure (pleura, peritoneum) is affected during an attack of herpes zoster."

Occasionally the sensation is impaired for a time over the affected area of skin, but it does not usually persist; and Head found that the impairment was generally most marked over the areas of deepest scarring; thermal sensations according to Head suffer more than others. The writer has observed several well-marked instances of "anæsthesia dolorosa" as a particularly obstinate sequela.

Diagnosis.—The diagnosis does not usually present any difficulty, but before the appearance of the eruption a pleurisy or pleurodynia may be closely simulated or in cases of herpes ophthalmicus a *tic douloureux.* The collections of grouped vesicles on erythematous bases with their definite lines of unilateral distribution usually form a characteristic picture; but sometimes doubts may arise from the lesions being abortive. Again, if the condition is seen for the first time when the vesicles have lost their early characters and run together into indefinite areas, the condition may resemble eczema, and the primary lesion may be further masked by secondary infections. Occasionally when the disease has occupied unusual regions, the erythema and vesicles have been mistaken for erysipelas.

Cases described as universal herpes zoster are undoubtedly instances of dermatitis herpetiformis (*q.v.*). In all cases of doubt a careful inquiry into the history of the onset and the accompanying symptoms will usually clear the matter up.

Treatment.—There is no marked method of treatment known which will certainly cut short the disease. The indications are to treat the patient on the same lines as for any other acute febrile affection, and to protect the affected parts from the air and from rupture. For this purpose the eruption may be freely dusted with zinc oxide and starch powder, and covered with a thick layer of medicated cotton wool and a bandage. The application of Unna's glyco-gelatin of zinc is often very efficacious, apparently arresting the eruption. When the pain is severe a small quantity of powdered opium added to the dusting powder will sometimes give relief. The application of camphor-chloral or belladonna liniment may also be useful.

Generally speaking, the use of ointments or greasy substances is unsuitable, but if the vesicles burst a boric ointment containing cocaine is valuable.

As soon as the attack is over, the patient should be given general tonics, such as strychnine, iron, quinine, or arsenic, with a generous diet and sound wine; while cod-liver oil is often of signal service. When the pain persists it becomes a very troublesome symptom; antipyrin, phenacetin, and other drugs of this class may be given, and the continuous current is frequently efficacious, but too often morphia is the only drug which gives any real or permanent relief.

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Hiccough.

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Treatment 7

Hiccough; Hiccup; Singultus. Hiccough is usually described as being produced by a sudden descent of the diaphragm, accompanied by a spasmodic closure of the glottis, the characteristic sound being due to the inrushing column of air striking against the closed vocal cords.

While there is no doubt as to the part that the diaphragm plays, the exact condition of the glottis is more open to question. Poore (1) considers that the assumption of a spasmodic closure of the glottis is not warranted, and it certainly seems difficult to understand why any such closure should occur, when it is remembered that during the descent of the diaphragm in ordinary respiration the glottis is dilated. It seems reasonable to suppose, therefore, that the closure during hiccup is only a *relative* one, due not to any spasm of the adductors of the larynx, but rather to a want of action of the dilators, the correlation between these latter and the diaphragm ceasing to exist when the diaphragm contracts irregularly and serves no useful part in respiration.

Ætiology.—Hiccough may be central or peripheral in origin. Irritation of the phrenic nerve in any part of its course may give rise to irregular diaphragmatic contractions, and in this way hiccough has been produced by tumours and other irritating lesions of the neck.

Most commonly, however, hiccough is a reflex act, the pneumogastric usually being the afferent nerve. Flatulence, overloading of the stomach, excess of alcohol, and the different forms of dyspepsia, are among the more common causes. It is also frequently associated with peritonitis, cardiac failure, uræmia, and other acute diseases, as typhoid fever, pneumonia, and pleurisy. Richardson (2) has suggested that its presence in acute diseases is frequently due to the alcohol which is prescribed. Tobacco smoking occasionally appears to be a cause. In an obstinate case recorded by Stevenson (3) large caseous masses were found after death at the root of each lung, which it was thought might have involved the phrenic or vagus nerves. In chronic cases recurrences are sometimes prone to take place at the menstrual periods.

Hiccough occasionally occurs as a true neurosis independently of any definite cause. In a case recorded by Ottoni (4), a patient, after suffering for some years from hysteria, suddenly became subject to the most obstinate hiccup, which, at the time the case was reported, had existed for thirty-four months, with rare intervals of ease, and it was always found that any temporary amendment was followed by a redoubled severity.

Liveing (5) mentions instances where the hiccup appeared to bear some relation to epileptic fits, attacks apparently occurring in place of a fit.

Symptoms.—The symptoms of hiccup are too well known to need any detailed description: the sudden inspiration, accompanied by the characteristic sound, is familiar to every one. Usually an attack causes only a slight temporary inconvenience, and either passes off spontaneously or is cured by some simple remedy. Occasionally, however, the condition is most obstinate, and the exhaustion produced by the constant repetition of the muscular efforts and want of rest become very serious, especially if the patient has already been weakened by existing disease. The length of time the symptoms last, and their severity, are very various. A case recorded by Foot (6) gives a good idea of the severity of the symptoms which can occur. The patient, a boy aged 15, hiccoughed incessantly, except when he was asleep, for twenty-six weeks. The onset of the attack was quite sudden, and he had previously had two other but much less severe attacks. The rate of hiccough was calculated on thirteen different occasions in this case. It varied from 8 to 22 per minute, and from 480 to 1320 per hour, the average being 14 per minute, or 840 per hour.

The significance of hiccough varies with its cause. Most commonly it signifies a slight digestive disturbance, and is of no importance. When it occurs during acute diseases it is usually a grave omen, especially in cases of peritonitis.

Treatment.—Ordinary attacks of hiccough either soon pass off spontaneously, or are readily amenable to some of the familiar remedies, such as holding the breath, sipping a glass of water, or taking several deep respirations at regular intervals. In cases which do not readily pass off, a draught containing aromatic spirits of ammonia or warm applications to the abdomen may be tried, or, if the stomach is obviously overloaded, an emetic may give the desired relief.

The more obstinate cases, unfortunately, often fail to yield to any of these remedies, and it would appear from records that no one drug can be relied upon in any given case. Among the preparations which seem to have been most successful in curing the complaint, nitro-glycerine, ether, turpentine, and liquid extract of ergot may be mentioned. Most observers are agreed that morphia exercises but little influence in checking the condition. Foot's case, which has already been referred to, recovered on pills of iodoform, extr. of Indian hemp, and extr. of conium. Under this treatment the hiccough diminished and finally ceased. Mechanical measures, such as blisters over the origin of the phrenic nerve and pressure upon its trunk at the root of the neck, and depression and traction of the tongue, have been practised, and the last-named method appears to have been successful in several cases which had failed to yield to medicinal treatment.

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Hill Diarrhœa. *See* SPRUE.

Hip-Joint.

A. Injuries of.

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INJURIES to the hip are numerous and may be serious, for even when they are caused by only slight violence the effects upon the patient are often far-reaching and may be of grave import.

BRUISED HIP.—Contusions of the hip, as a result of falls or blows, are usually of importance only in children and in old people. In children because they may form the starting-point of tuberculous or other infective disease of the joint: in old people because a contusion of the hip often resembles in its symptoms, though never in its signs, a fracture of the neck of the femur. But a fall or repeated blows upon the great trochanter may lead at any age to enlargement of the bursa beneath the insertion of the gluteus maximus, or to a remarkable form of chronic osteoporosis known incorrectly as "interstitial absorption of the neck of the femur."

Symptoms.—In its simpler forms the symptoms of a contusion of the hip differ but little from those of a bruise elsewhere. There is tenderness over the part struck, followed in a few hours by swelling and stiffness, which slowly disappear, whilst the skin passes through a succession of colours due to the oxidation changes produced by the tissues on the hæmoglobin of the extravasated blood. In old people the shock of the injury is sometimes sufficient to paralyse the limb for a time. The thigh lies motionless on its outer side and slightly abducted, whilst the upper part of the thigh is swollen and tender. It is by no means easy to ascertain in these cases whether the patient is suffering from a bruise or from an intracapsular fracture of the femur. In a bruise the thigh may seem to be slightly lengthened, and the great trochanter maintains its normal relation to the anterior superior spine of the ilium; whilst in a fracture there is some shortening, and the trochanter is displaced and no longer rotates round its proper axis. Even these guides, however, are lost in a patient who suffers from osteoarthritis, and an attempt must then be made to establish the diagnosis by means of a skiagraph. The prognosis of a bruised hip is good, but the practitioner should warn an old person or the mother of a delicate child of certain special risks, which will be described immediately.

Treatment.—The treatment in the simpler forms consists of a few days' rest in bed, during which the hip may be rubbed with an embrocation or may be massaged by a skilful shampooer. When the shock is so great as to make it doubtful whether the injury is a bruise or a fracture the patient must be treated as though he were suffering from an intracapsular fracture.

After-effects.—The pain sometimes continues for a long time, and is increased by exercise after a severe bruise upon the outer side of the thigh in the neighbourhood of the great trochanter. At first there is no shortening, but the patient limps more as the local pain and discomfort diminish, until the injured limb is found to be appreciably shorter than its fellow when the thighs are measured from the anterior superior spines of the ilia to the adductor tubercles. The condition occurs most often in old people, but it is also met with occasionally in young adults who have apparently been healthy until they met with the accident. The pathology is in dispute, and the condition has been described hitherto under the vague expression "interstitial absorption of the neck of the femur." Mr. Arbuthnot Lane has lately advanced good reasons for assuming it to be a result of severe bruising of the cartilages covering the upper part of the head of the femur and the corresponding portion of the acetabulum. The injured cartilages and the underlying bone become absorbed as a result of the chronic inflammation thus set up. The head of the femur becomes altered in shape, and the acetabulum becomes deeper and larger in a manner similar to that seen in osteoarthritis. The great trochanter seems, therefore, as though it had approached the head of the femur, but in reality the trochanter comes nearer to the rim of the acetabulum because the head of the bone lies deeper in its socket, whilst the length of the neck of the femur remains unaltered.

The prognosis is unsatisfactory, especially if the patient is not seen until there is well-marked shortening.

The treatment in the early stages consists in keeping the patient in bed, with a weight and extension applied to the injured limb to remove all pressure from the joint, and to prevent, as far as possible, the continued absorption of the bruised cartilage and bone. When the shortening has commenced, the difference in the length of the legs should be accurately adjusted by a thick-soled boot, for the limping tends to increase the absorption.

Bursal Enlargements.—Blows on the outer side of the hip, injuries, exposure to cold, and certain occupations, such as tailoring, which involve prolonged sitting in bad attitudes, may lead to pain and swelling over the top of the great trochanter. After a longer or shorter time a well-defined swelling appears in the long axis of the limb. The swelling is limited abruptly by the femoral insertion of the gluteus maximus, and is due to an enlargement of the bursa beneath the aponeurosis of this muscle. If it be left untouched it opens at the outer side of the thigh below the level of the great trochanter. In unhealthy people it often suppurates and tracks far and wide, leaving most troublesome sinuses, but it is not often associated with any necrosis of the bone even when there has been extensive suppuration.

Treatment.—The treatment consists in laying open the bursa freely—by cutting through the fascia lata parallel to its fibres—turning out its contents, breaking down all bridges of connective tissue, scraping the wall with a flushing scoop, through which a stream of boracic lotion at a temperature of 110° F. is passing, and afterwards suturing the edges of the wound and obtaining union by first intention. A drainage-tube may be inserted at the time of the operation if it be removed twenty-four hours later. The bursa sometimes communicates by a narrow channel with a small subcutaneous abscess, forming a "collar-stud" abscess, and it is important in dealing with such an abscess not to overlook the point of communication between the small subcutaneous abscess and the larger one situated beneath the fascia lata. The bursa beneath the the ilio-psoas tendon (Fig. 1, 10) may also become enlarged as a result of injury or from infection with syphilis, rheumatism, or gonorrhœa. It forms a smooth and fluctuating swelling, which lies below Poupart's ligament, and may extend downwards as far as the middle of the thigh. The swelling may cause neuralgic pains from pressure upon the nerves, or venous thrombosis from pressure upon the deep veins. The movements at the hip are often hampered, and the limb may

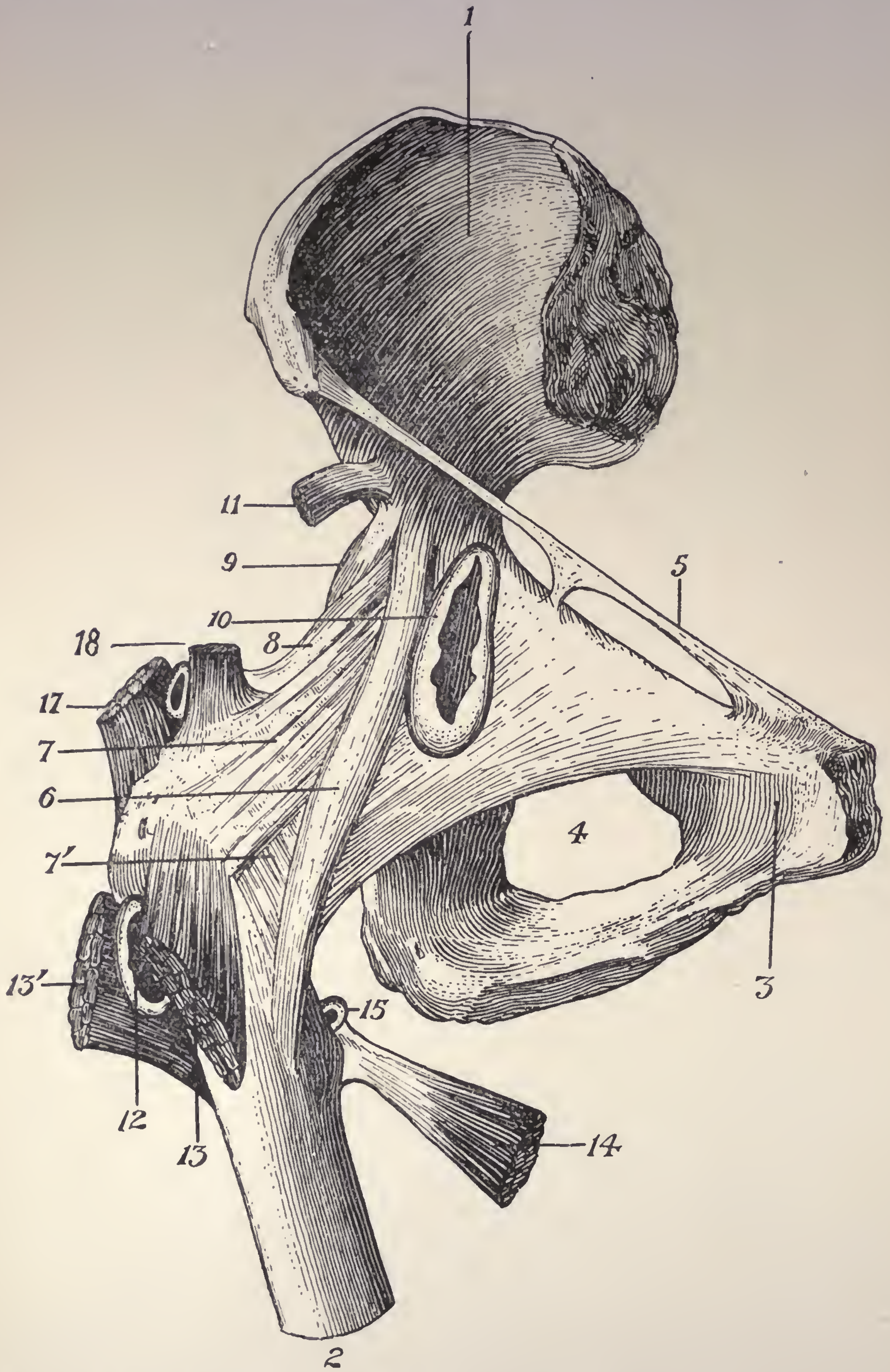

Fig. 1.—Front view of the Hip-Joint. 1. Ilium; 2. Femur; 3. Pubis; 4. The thyroid or obturator foramen; 5. Poupart's ligament; 6, 7, 7′, 8, and 9. The capsular ligament of the hip, divisible into:—6. The inner branch or fasciculus of the ilio-femoral, or Y-shaped ligament of Bigelow; 7. The outer branch or fasciculus of the Y-shaped ligament of Bigelow; 7′. The intermediate portion of the capsular ligament; 8 and 9. The upper portions of the capsular ligament; 10. The bursa lying between the ilio-psoas and the capsule, of the joint; 11. The straight head of the rectus femoris cut short; 12. The bursa separating the vastus externus (13) from the gluteus maximus (13′); 14. The tendon of the ilio-psoas cut short; 15. The bursa lying between the ilio-psoas tendon and the lesser trochanter; 16. A small bursa is also shown lying between the insertions of the gluteus medius (17) and minimus (18). (Copied from Testut's *Traité d'Anatomie humaine.)*

become slightly flexed, abducted, and rotated outwards. But it is not difficult to distinguish an enlarged ilio-psoas bursa from hip disease, because there is neither shortening, rigidity, nor tenderness of the joint. The treatment consists in freely incising the bursa after it has been exposed through a skin incision. It is absolutely necessary that the wound should heal by first intention. There are other bursæ in the immediate neighbourhood of the hip-joint situated as is seen in Fig. 1, at the insertion of the ilio-psoas tendon, between the vastus externus and the gluteus maximus, and between the gluteus medius and minimus. I have seen them all enlarged in different persons chiefly as a result of tuberculous disease or osteoarthritis.

Sprained Hip.—Sprains of the hip are generally the result of sudden extension of the thigh combined with abduction, and are rarely caused by forced flexion. They affect the hip in more than one way. Sometimes a few fibres of the pectineus, the adductor brevis, or the adductor longus muscles are torn: in other cases the capsular ligament is wrenched and a little synovitis ensues: whilst in others, again, the ligamentum teres with the fatty tissue at its base is said to be nipped beneath the transverse ligament of the joint. The latter accident happens when a patient slips with the thigh forcibly abducted and at the same time slightly flexed. The thigh becomes everted, abducted, and apparently lengthened, whilst its movements are greatly hampered by the reflex spasm of the surrounding muscles. Flexion followed by rapid extension of the hip sets free the ligamentum teres and restores immediate use to the limb, which should then be kept at rest for a few days. Partial rupture of the muscles and slight inflammation of the synovial membrane are usually amenable to rest in healthy people, but in unhealthy persons they often give rise to much trouble. There may be lameness without any impairment of movement in the joint, perhaps from a little interstitial myositis or from slight teno-synovitis after partial rupture of the muscles or injury to the tendons and their sheaths, or if the synovitis of the hip has been plastic rather than serous, it may have caused local adhesions and thickening of the capsule. These conditions often lead the patient into the hands of the bone-setter, who rightly believes the mischief to be outside rather than inside the joint when there is an absence of pain on direct pressure over the part, or when shocks are impressed upon the heel, particularly if pain is felt upon using the heel as a pivot for the body and when it is produced by stooping forwards. Passive movements and the breaking down of the adhesions, with or without an anæsthetic, are of great service in some of these cases, especially if the surgeon take care to fix any painful spot by pressing his thumb upon it at the instant when the forcible movement is made.

Wounds.—Wounds of the hip may so injure the surrounding tissues, and even the capsule, as to lead to serious impairment of the movements of the joint; or they may cause a secondary arthritis; but the joint itself is so thoroughly protected from injury that it is hardly ever injured, except as a result of gunshots. The swelling of the capsule forms the most certain sign of injury, either at once, as a result of the effusion of blood, after a short interval from an increase in the synovial fluid, or quite late from suppuration. The swelling is felt easily, and may so raise the femoral artery as to make the pulsations feel subcutaneous. The particular dangers of the injury are liability to suppuration and implication of the pelvic organs. The difficulty of rendering the joint immobile forms a great obstacle to successful treatment, and it is often necessary, therefore, to excise the head of the femur either immediately after the injury or at a later period. The experience gained in the South African war, however, shows that the present gunshot wounds of joints are by no means so serious as those inflicted in previous campaigns. A bullet may pass completely through a joint without leading to permanent impairment of its function.

Dislocations of the Hip are amongst the rarer forms of injury met with in general surgical practice. They are the result of considerable violence, and nearly always occur in adults, though most surgeons have seen a few cases of true traumatic dislocation in children: in old people a fracture of the neck of the femur usually takes the place of dislocation of the hip.

Pathology.—An anatomical examination of the hip shows that dislocation of the head of the femur takes place more easily in some postures than in others, and at the same time it explains why the head of the bone generally takes up certain fixed positions after it has left the acetabulum. The capsule of the hip is strengthened in front by a fibrous expansion—the ilio-femoral or Y-shaped ligament of Bigelow—whilst behind is the ischio-capsular ligament, extending from the back of the tuber ischii to the trochanters. Of these ligaments the ilio-femoral (Fig. 1, 6, 7, 7′) is the more important. It consists of a thickened portion arising from the front of the ilium just below the anterior superior spine, and passing downwards to be inserted into the whole length of the anterior intertrochanteric line; its base extends, therefore, from the front and upper part of the great trochanter obliquely across the front of the femur to just below the lesser trochanter. The outer and the inner parts of the ligament are thicker than the intermediate portion, so that the front part of the ligament consists of two denser fasciculi—the outer and inner branches of the Y-ligament—connected together by a less dense layer. At the back of the joint, in addition to the ischio-capsular ligament, the capsule is strengthened by numerous tendons and muscles (Fig. 2), some of which are in very intimate relation with the capsule, whilst others, from the nature of their tendons, are veritable ligaments. The tendon of the obturator internus muscle (Fig. 2, 5) is one of the most important of these structures, because it forms a compact and well-defined band of fibrous tissue lying immediately over the middle of the head of the femur. Of late years, however, somewhat too great an importance has been attached to it in connection with dislocation of the hip backwards, for it is by no means unusual to find the obturator internus torn.

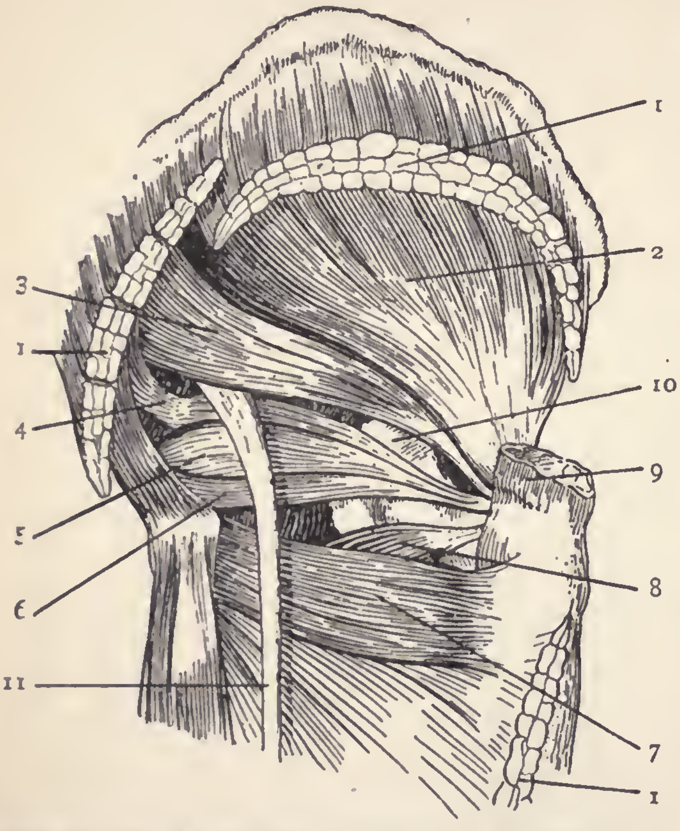

Fig. 2.—The Anatomical Relations of the Hip-Joint. 1. The gluteus maximus cut short; 2. The gluteus minimus; 3. The pyriformis; 4. The gemellus superior; 5. The obturator internus; 6. The gemellus inferior; 7. The quadratus femoris; 8. The obturator externus; 9. The insertion of the gluteus medius; 10. The head of the femur (the capsule of the joint is not shown); 11. The great sciatic nerve. (From Helferich's *Fractures and Dislocations*, translated by Jno. Hutchinson, Esq., Junr. The New Sydenham Society's ed.)

Primary Dislocations.—The capsule is thinnest and least protected opposite the notch in the acetabulum, which is situated at the bottom of the joint and a little behind its vertical axis. The acetabulum too is shallow and incomplete at this spot. When the thigh is flexed and abducted the head of the femur presses against this weak part of the joint, and at the same time the ligamentum teres is fully relaxed. A sudden and violent movement imparted to the body of the femur when the thigh is thus bent and abducted may drive the head of the bone out of the socket through a rent in the capsule, the head being displaced downwards and a little backwards, whilst the rent in the capsule varies in size from a minimum equal to the diameter of the head to a laceration involving fully two-thirds of circumference of the acetabulum. This seems to be the usual method by which a dislocation of the femur is produced so that the common primary dislocation is downwards and inwards. But in some cases a direct dorsal dislocation may be brought about by the application of force to the adducted and flexed thigh. The head of the bone then escapes above the tendon of the obturator internus, if the flexion has only been moderate, below it if extreme. A portion of the rim of the acetabulum is often torn off in direct dorsal dislocation, and in every dislocation a continuance of the force producing the injury may lead to fracture of the pelvis or of the femur. Many of the muscles surrounding the joint (Fig. 2) may be torn, either directly (*e.g.* the obturator externus, quadratus femoris, obturator internus, or pyriformis) by the head of the bone, or, as in the case of the gracilis and adductor longus, by overstretching when there has been extreme abduction. Injury to the great sciatic nerve may lead to neuritis, to partial paralysis, or even to rupture of the nerve.

Secondary Dislocations.—Although in most cases the first position assumed by the head of the bone after it escapes from the acetabulum is downwards, subsequent movements of the injured limb cause this to be only temporary, and the head passes either outwards or inwards, the final position being dependent upon the remnant of untorn capsule binding the shaft of the femur to the rim of the acetabulum. From the character of the capsule the Y-ligament most often remains untorn, and when this is the case, or when it is only slightly injured, the head of the bone passes into certain well-recognised positions, forming a series known as the regular dislocations; but when the Y-ligament (Fig. 1, 6, 7, 7′) is completely or in great part destroyed, the head of the bone may stray into quite unusual places, and a series of irregular or anomalous dislocations is then produced. It should be remembered that although the classification here adopted is useful for clinical purposes, and for guidance in the reduction of the majority of cases of dislocation of the hip, it is by no means pathologically accurate, and it is for this reason that in some cases the ordinary rules for reduction by manipulation fail absolutely.

Regular Dislocations.—In the regular dislocations the head of the femur passes outwards, resting upon the ilium either above the tendon—*the dorsal dislocation* of the older surgeons (Fig. 3, *1*)—or below the tendon of the obturator internus—*the sciatic dislocation* (Fig. 3, *3*). In other cases the head of the femur passes inwards, either inwards and downwards (Fig. 3, *2*) —*the thyroid or obturator dislocation*—or inwards and upwards (Fig. 3, *4*) —*the pubic or subspinous dislocation*—when the head of the bone lies upon the horizontal ramus of the pubes just internal to the anterior inferior spine of the ilium. All inward dislocations present the same general characteristics of abduction of the thigh with rotation outwards, and with a single exception, all outward dislocations cause the thigh to be adducted and rotated inwards. The exception is *the anterior oblique dislocation* when the thigh is everted and crosses the opposite one high up, the toes being turned outwards, the head of the bone lying on the dorsum ilii just behind the anterior inferior spine. The Y-ligament is intact, and the dislocation may be converted into the ordinary dorsal form, in which the thigh is inverted by inward circumduction of the extended limb across the symphysis with inward rotation.

When the outer branch of the Y-ligament (Fig. 1, 7) is torn, the head of the femur may pass forwards and upwards until it lies at a higher level than when the ligament is intact—*a supraspinous dislocation* is then produced—the limb being much shortened, slightly abducted and everted. An anterior oblique dislocation is readily converted into a supraspinous dislocation by rupture of the outer branch of the Y-ligament, so that this variety may be produced in two ways, either by the head passing outwards and upwards or inwards and upwards. When the outer branch of the Y-ligament is torn and the head of the femur passes outwards and backwards, *an everted dorsal dislocation* is produced, the limb being everted and slightly abducted, so that it resembles a fracture of the neck of the femur. Flexion, adduction, and internal rotation of the thigh readily convert an everted dorsal into the ordinary inverted dorsal dislocation.

When the inner branch of the Y-ligament (Fig. 1, 6) is torn, the head of the bone may slip much farther forwards than is usual, and the head may lie upon the pubes close to the pubic spine.

Anomalous Dislocations.—Extensive destruction of the Y-ligament enables the head of the femur to pass upwards or downwards, either vertically below the acetabulum, backwards upon the tuber ischii, or downwards and forwards into the perineum.

Relative Frequency.—The regular dislocations are said to occur in the following order of frequency :—

50 to 55% dorsal above the tendon of the obturator internus.
20 to 25% sciatic or dorsal below the tendon of the obturator internus.
10 to 15% thyroid or obturator dislocations.
5 to 10% pubic or subspinous.

Signs.—The general signs attending a dislocation of the hip are an alteration in the length of the injured limb, diminished power of movement, with locking of the joint in certain positions, and an alteration in the axis of the limb. In a simple dislocation there is no crepitus, and when the displacement is reduced there is no tendency for the signs to reappear. But it is by no means unusual to find extensive fractures of the pelvis or femur associated with dislocation of the hip, and then, as well as when a portion of the rim of the acetabulum is separated, there will be both crepitus and a tendency for the dislocation to recur after reduction.

The hip is wider and flatter than its fellow in a dorsal dislocation (Fig. 3, *1*) and the injured limb is considerably shorter. The thigh is slightly flexed, adducted, and rotated inwards, so that when the patient is erect the great toe rests upon the instep of the opposite foot, and the knee touches the patella of the uninjured side near the upper margin. The great trochanter lies some distance above Nélaton's line—drawn from the anterior superior spine of the ilium to the most prominent part of the outer lip of the tuber ischii—and it lies unduly near the iliac spine because the head of the bone is directed backwards upon the dorsum of the ilium. The femoral vessels have lost their support owing to the displacement of the head of the femur, and the groin appears therefore to be unduly deep. The head of the femur can generally be felt in its new situation, but all the movements of the joint are greatly hampered. Flexion is most free, adduction is difficult abduction is impossible. When the head of the bone lies below the tendon of the obturator internus (Fig. 3, *3*) there is less shortening of the limb, but the flexion, adduction, and inversion are somewhat more marked than in the dorsal form.

Method of reducing a Backward Dislocation of the Hip.—Reduction is brought about by manipulation, which should be undertaken immediately; but if the patient be not seen until some time after the accident, it is better to defer all manipulation until an anæsthetic can be given, and the attempt made under the most favourable conditions of assistance and position.

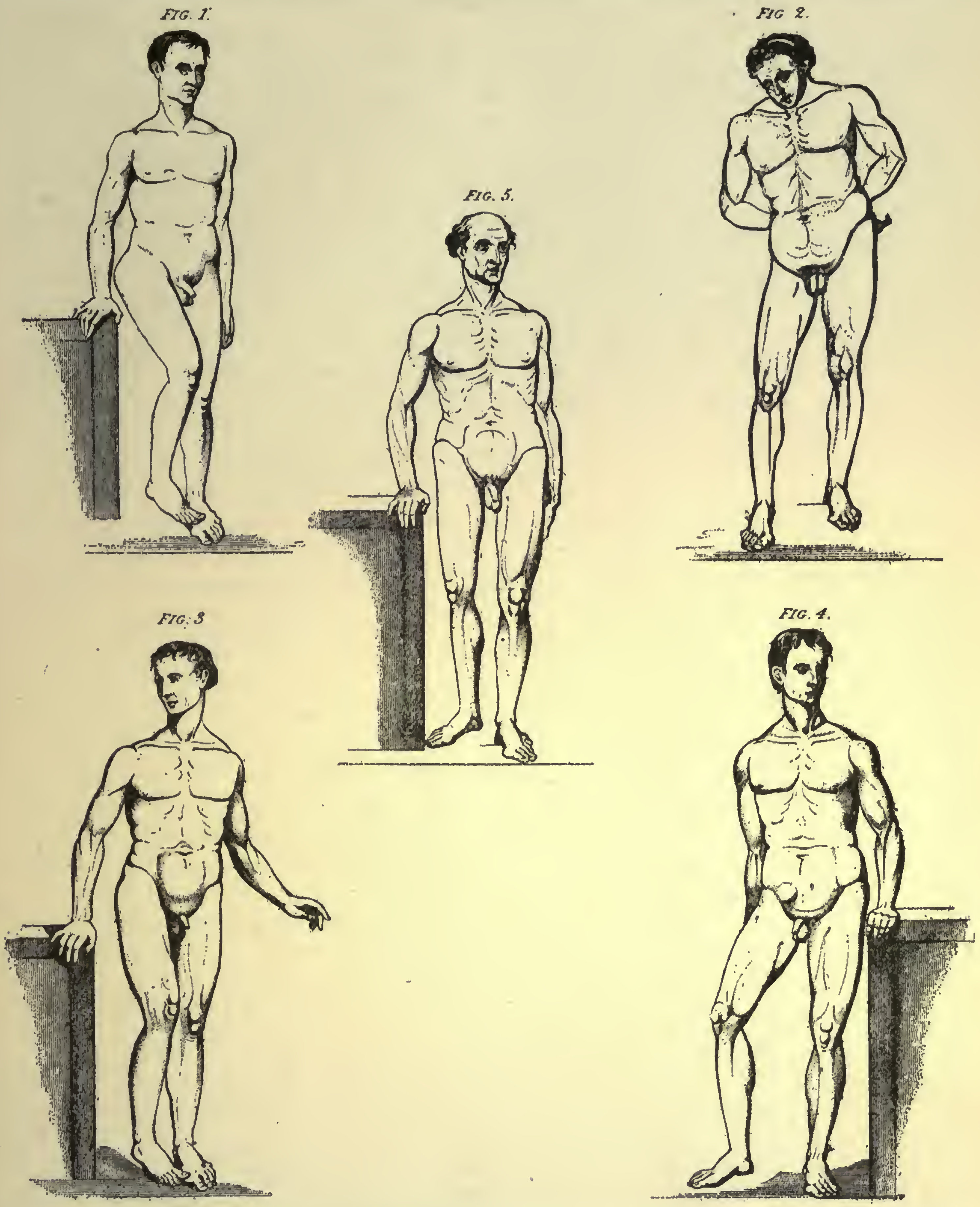

Fig. 3.—Sir Astley Cooper's diagrams to show the positions of the limb in the different dislocations of the thigh-bone, and in intracapsular fracture of the neck of the femur. *Fig. 1.* The right thigh-bone dislocated upwards upon the dorsum ilii. *Fig. 2.* Dislocation of the right femur downwards into the thyroid or obturator foramen. *Fig. 3.* Sciatic dislocation of the right hip. *Fig. 4.* Pubic or subspinous dislocation of the right thigh. *Fig. 5.* Intracapsular fracture of the neck of the right femur.

The patient lies flat on his back upon a mattress placed on the floor, whilst the surgeon kneels beside him and grasps the leg just above the ankle with one hand whilst he places his hand, or, if the thigh be heavy, hooks his elbow below the ham of the injured limb. He then flexes the leg upon the thigh to relax the ham-string muscles, and the thigh upon the pelvis to relax the Y-ligament or other portion of untorn capsule, and to bring the head of the femur below the acetabulum. This position of flexion with slight adduction is to be maintained for a few seconds, care being taken not to flex the thigh too much lest the head of the bone pass too far forwards; the thigh is then fully abducted, rotated outwards to its utmost extent, and brought down to the couch in an extended position. The movement of abduction brings the head of the femur from below and behind the acetabulum to the rent in the capsule, the external rotation still further raises the head and causes it to pass through the rent, whilst the extension replaces it at the bottom of the acetabulum.

If reduction does not take place the head of the bone will pass inwards and the dorsal dislocation will be converted into a thyroid or subspinous displacement. The surgeon need not be discouraged, but he should repeat the manipulation, and at the same time he should cautiously increase the amount of adduction and inversion, whilst he diminishes the amount of flexion. He must never allow his efforts to become violent lest he fracture the femur, and he should remember that each repetition of the manipulation is likely to lead to further tearing of the soft tissues around the joint. When the rent in the capsule is small, as is shown by the restricted movements of which the limb is capable, circumduction will sometimes restore the head of the bone to its socket, for the capsule is then so short that the movement compels the head to hug the rim of the acetabulum until it re-enters the cavity. The circumduction is effected by sweeping the leg and thigh round in a wide circle, the patella being always kept to the front, for the articular surface of the head looks nearly in the same direction as the internal condyle of the femur. This method of manipulation, however, is open to two serious objections. It happens sometimes, after an apparently successful reduction of a dorsal dislocation by free circumduction, that the knee remains bent whilst the thigh continues flexed on the pelvis and is abducted and rotated outwards. This want of freedom is due to the fact that the sciatic nerve is stretched over the front of the neck of the femur, lying first beneath the inner portion of the capsule and the tendon of the psoas, and then descending through the rent in the muscular wall of the thigh to the adductor magnus. A second objection to the method of reduction by circumduction is that a part of the capsule may become folded over the head of the bone and may thus prevent reduction.

In the thyroid or obturator dislocation (Fig. 3, *2*) the injured thigh is longer than the other, though too much reliance must not be placed upon this sign; it is abducted and flexed. The hip is flattened and the great trochanter is less prominent than normal. The toes point forwards. When the head of the bone lies in the perineum there is marked flexion and abduction of the thigh.

Reduction is accomplished by placing the patient supine as before. The surgeon then flexes the leg on the thigh, the thigh on the abdomen, adducts, inverts, and brings the thigh downwards in extension. This form is usually easier to reduce than the dorsal displacement.

In the pubic dislocation (Fig. 3, *4*) the hip is flattened and the great trochanter lies above Nélaton's line and nearer to the symphysis pubis than is natural. The thigh is shortened, abducted, rotated outwards, and slightly flexed. The femoral artery is displaced inwards and the head of the bone can be felt in its new position. This form of displacement is sometimes attended by great pain in the course of the anterior crural nerve.

Reduction is brought about in the same manner as in the obturator variety by flexing the leg on the thigh, the thigh on the abdomen, then adduct, invert, and bring down in full extension.[1]

The after-treatment of a reduced dislocation consists in keeping the patient in bed for a fortnight, the injured limb being placed between sandbags or bandaged to a long outside splint. Passive movement may then be employed, but the patient should not be allowed to walk about for at least six weeks after the accident, though he may be permitted the use of crutches. The duration of treatment in a case of dislocation complicated by fracture will necessarily be prolonged, and a weight and extension should be applied.

The prognosis after reduction will depend upon the nature of the injury and the ease with which the displacement has been reduced. It is good in simple cases, but the patient should be warned that the accident may recur, that there may be some permanent lameness from injury to the muscles, that there may be some neuritis, and that it may form the starting-point of disease of the joint.

The obstacles to reduction are numerous and are sometimes insuperable. One of the most common appears to be due to the fact that the capsule has been torn from the neck of the femur, so that it hangs as a covering over the acetabulum and prevents the head re-entering the socket: in other cases part of the obturator externus is torn, and the muscular fibres are pushed before the head into the acetabulum, whilst in yet other cases the head of the femur may lie as it were "in chancery" between the tendons of the pyriformis and the obturator internus muscles (Fig. 2, 3 and 5). When the capsule has been pushed into the socket from the dorsal aspect it may sometimes be removed by flexing and abducting the thigh, whilst if it has entered from the thyroid aspect flexion and adduction of the femur may displace it. After engaging the inverted capsule the femur should be rotated inwards to tighten the Y-ligament and drive the head well into the acetabulum, whilst at the same time the knee is raised and the capsule or muscle is thus removed from its cavity.

The irreducibility in long-standing dislocations is due to causes other than those just mentioned, though the capsule again plays an important part. When the cavity of the acetabulum remains unoccupied the torn capsular ligament falls over it, and the stump of the ligamentum teres undergoes inflammatory changes. A proliferation of connective tissue cells takes place, and the socket becomes occupied with connective tissue which is at first soft, but soon becomes dense and fibrous. If the rent in the capsule is only a small one it often heals early, and has been found completely repaired eighteen days after the dislocation. The ligament teres, too, shows remarkable powers of repair, and a new and very complete joint may be formed at the place where the head lies in its new position. The shortening of the muscles and tendons inserted into the upper end of the femur and fibrous adhesions, or a new capsule formed about the head, greatly militate against the successful reduction of old-standing dislocations of the hip.

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[1] Mr. Sturrock has recently (*The British Medical Journal*, i. 1900, p. 845) reduced two regular dislocations of the hip by the following manipulation: The patient being anæsthetised and laid upon his back on the floor, the surgeon kneels upon his left knee when the left hip is dislocated, and on the left side of the patient. The patient's thigh is then carefully flexed to a right angle, and while this is being done the leg is also flexed to a right angle and laid with the most prominent part of the calf on the right knee of the surgeon. The ankle is then firmly grasped with the left hand and the condyles of the femur with the right. The thigh is then abducted for thyroid dislocations, adducted for dorsal and pubic, and rotated inwards for all varieties, by drawing the foot away from the middle line and keeping the knee steady. Traction is now made by steadily depressing the ankle, the surgeon using his knee as the fulcrum: the patient's leg makes a most powerful lever. The thigh is then rotated outwards and, while this is being done, the head of the femur slips into the acetabulum.

The treatment of unreduced dislocations of long standing is one which may require the most anxious consideration of the surgeon. It consists necessarily in leaving the patient alone, in subcutaneous division of the contracted muscles and tendons, in osteotomy, in arthrotomy, or in excision of the head of the femur. When the inconvenience is slight and the dislocation of long standing it is generally wise to leave the patient alone, but when he is crippled and in pain, arthrotomy and excision of the head of the bone seem at present to offer the most satisfactory results. Arthrotomy consists in cutting down upon the acetabulum by an incision planned to permit easy access to the great trochanter. In the majority of cases this will be found to be in front of the joint along a line separating the posterior border of the tensor fasciæ femoris from the gluteus medius. The head and neck of the femur are then laid bare, and all the muscular attachments are divided as low as the lesser trochanter, until the whole upper end of the bone, including the great trochanter, is absolutely free, the division being subperiosteal as far as is possible, and the proximity of the great sciatic nerve not being overlooked. The acetabulum is then excavated and enlarged sufficiently to allow the head of the femur a ready entrance. The head is replaced in the socket thus prepared to receive it, the wound is closed, extension is applied, and the limb is fixed for at least a month in a position of slight abduction. Excision should only be performed when arthrotomy has failed, or is manifestly impossible, for though it gives the patient a useful limb it also gives him a shortened one. When an excision has been decided upon, therefore, as little of the neck as possible should be removed.

Other difficulties may also arise in connection with a dislocation of the hip, and chief amongst these are the cases where the dislocation is compound or when the femur is broken in the upper third. In the less serious cases of compound dislocation the wound and the surrounding skin should be thoroughly cleansed, and the head of the bone should then be guided into the socket, the wound being enlarged if it be necessary, but more often it will be necessary to excise the head of the bone. Amputation is so fatal an operation that it should be avoided except as a very last resource. But when it has to be done, the operation should not be delayed until inflammation and suppuration have made all the tissues highly vascular, as hæmorrhage is the chief danger of this operation. When the dislocation is complicated by a fracture, an incision must be made along such a line as will enable the head to be replaced and the fracture to be united with stout silver wire. The bulk of the soft tissues covering the injured hip will sometimes render impracticable any operation, and the surgeon must then be content to leave the dislocation unreduced and secure union of the fracture. The subsequent treatment will depend upon the amount of movement which the patient obtains.

Epiphyseal Separations.—*The anterior inferior spine of the ilium* has attached to it the straight head of the rectus femoris muscle, which is rendered taut when the hip is extended, the reflected head being tightened when the rectus is used to extend the knee during flexion of the hip. Separation of the epiphysis of the anterior inferior spine of the ilium is the result of muscular force transmitted through the straight head of the rectus, and has occurred in young adults who were just bounding off at the beginning of a race, when they had been standing with one leg advanced and bent both at the hip and knee whilst the injured leg was straight. The symptoms were sudden but not very severe pain, flexion at the hip and knee with crepitus, sometimes difficult to obtain, as only a scale of bone may be detached. The treatment consists in the application of a plaster of Paris spica for a month. The prognosis is good.

The epiphyseal head of the femur may be separated at any time after the age of 4 years, though the accident is most common between the ages of 14 and 18 years. It is probable that in early infancy the whole upper end of the femur may be detached from the shaft, but in older children it is an intracapsular separation of the cartilaginous head. The accident happens as a result of direct injury to the outer side of the thigh below the great trochanter when the leg is adducted and semiflexed. The injured limb is everted owing to the weight of the leg, and there is slight flexion with abduction or adduction. The eversion is sometimes replaced by inversion, due perhaps to the lower fragment being placed in front of the upper part of the bone. The great trochanter is more prominent, and lies higher on the injured than on the uninjured side, and when the separation is complete there is muffled crepitus with very considerable shortening. The amount of movement varies with the separation of the fragments; it is usually slight, but when the periosteum remains untorn the patient may retain considerable power over his leg, for the periosteum covering a growing bone is relatively thicker and stronger than that covering a mature bone. The pain varies greatly, but it seems to be somewhat less severe than in a fracture of the neck of the femur.

The injury has to be distinguished from a traumatic and from a congenital dislocation, from a fracture of the neck of the femur, from hip disease, and from a simple bruise. The Röntgen rays help materially in the diagnosis.

The prognosis is generally good. Bony union occurs unless wide separation with laceration of the periosteum leads to non-union, an unfortunate result, which is most likely to occur in children who have been allowed to move the limb unrestrictedly in consequence of failure to recognise the nature of the injury. Some impairment of growth may take place in the upper third of the femur, leading to alterations in the head and neck of the bone, simulating the rachitic deformity known as coxa vara (see p. 24).

The treatment consists in the application of a weight and extension for three weeks, after which the patient is allowed to go about on crutches either in a Thomas splint or with a poroplastic splint fitted round the pelvis and extending down the outer side of the thigh. The prognosis is good though the repair is sometimes slow.

The epiphysis of the great trochanter may be separated between the thirteenth and sixteenth years of age generally by direct violence, as by a fall on the hip or a blow on the great trochanter, though the injury is not necessarily severe. In many cases the displacement is slight, but when the separation is complete the epiphysis is drawn upwards and somewhat backwards and inwards by the glutei and rotator muscles. Crepitus may or may not be felt, but there is always pain, swelling, and extravasation over the seat of injury. The prognosis is by no means good, for several of the recorded cases have died of septic poisoning. A long external splint, interrupted at the hip, should be applied as soon as possible, and the patient must be carefully examined from day to day to discover whether he has any symptoms of infective osteomyelitis. If necessary an incision should be made down to the bone before an abscess forms.

FRACTURES OF THE HIP.—*The acetabulum* may be broken either through its cavity or along the rim. A fall upon the trochanter may cause a simple fissure extending across the socket, it may split the acetabulum into its three constituent parts, or it may drive the head of the femur through the base into the pelvis. Fissured fractures give rise to few symptoms besides pain and lameness, and even when there has been considerable displacement of the fragments in the more severe fractures most of the recorded cases have been diagnosed as examples of fracture of the neck of the femur. Such errors will probably be less frequent in the future, as the Röntgen rays can be utilised when there is any doubt as to the nature of the injury. When the head of the bone is driven into the pelvis, pressure upon the great trochanter causes deep-seated pain, and there is often evidence that the pelvic viscera, especially the bladder, have sustained serious injury.

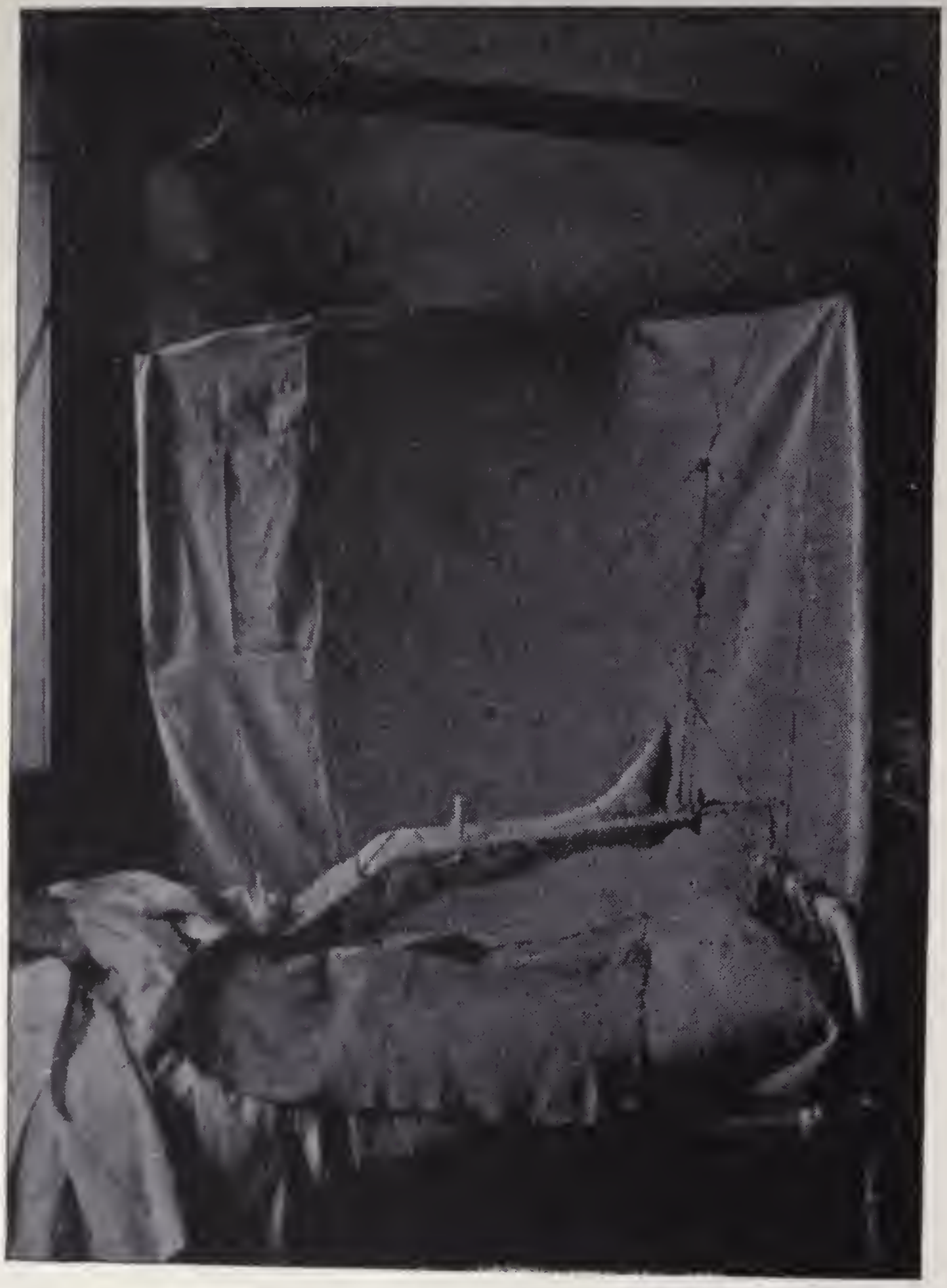

Fig 4.—A photograph of a case of fractured femur treated by a Hodgen splint to show the method of suspension. The details of the splint may be seen in vol. iv. p. 33. (From a photograph by Mr. G. S. Wynne of St. Bartholomew's Hospital.)

The prognosis of fissured fractures of the acetabulum is favourable, but when the head of the bone has been driven through the base the injury often proves fatal, either at once from shock or after a time from prolonged suppuration and septic absorption.

In the simpler forms of injury the treatment consists in keeping the patient at rest with a pelvic band and a weight and extension on the injured side. This may be replaced by a plaster of Paris spica at the end of six weeks, after which the patient is allowed to go about on crutches.

Fractures of the rim of the acetabulum are usually associated with direct dorsal dislocations of the femur. The symptoms are those of a dislocation of the hip backwards with the addition of crepitus and a great tendency for the displacement to recur after reduction. The prognosis is not very good since permanent lameness may occur from the impossibility of keeping the head of the femur in the acetabulum. The treatment consists in reducing the dislocation, and afterwards applying a Hodgen's splint (vol. iv. p. 33 and Fig. 4) for a month or six weeks after the accident.

Fractures of the Upper End of the Femur.—Although *intracapsular fractures* of the neck of the femur generally occur in persons over 60 years of age, they are undoubtedly met with as rare accidents in children in consequence of injuries of the same nature but of somewhat greater severity than those causing a separation of the epiphysis. The fracture is common in old people, because the changes which occur in the bones as a result of increasing age are especially marked in the upper third of the femur. These atrophic changes lead to an enlargement of the cancelli in the neck of the femur, whilst the compact layers of bone undergo a process of absorption which renders them particularly liable to yield when they are subjected to a strain or twist. The osteoporosis is especially marked in the denser layer of bone which reaches from the under part of the head to the lesser trochanter—known as the *calcar femorale* of Merkel. In women, too, the neck of the femur has to withstand a greater strain in supporting the weight of the trunk than in men, for in women the width of the haunch-bones causes the neck of the femur to form with the shaft an angle which is nearer a right angle than it is in the male pelvis.

An intracapsular fracture, therefore, often occurs in persons over 60 years of age, and in women more often than in men. Falls upon the feet or knees, and blows upon the front of the great trochanter, are particularly likely to produce an intracapsular fracture, and it is often remarkable how slight an injury, especially if a twisting force be applied, is sufficient to cause it. The signs (Fig. 3, *3*) are eversion of the limb with slight shortening. The great trochanter is less prominent, lies higher, and is situated more posteriorly on the injured than on the uninjured side. In a few cases the eversion is replaced by inversion. Crepitus may be obtained by drawing the thigh downwards and rotating it inwards, but it is not usually well marked, nor is it necessary to obtain it. The fascia lata is relaxed (Fig. 5) between the great trochanter and the crest of the ilium

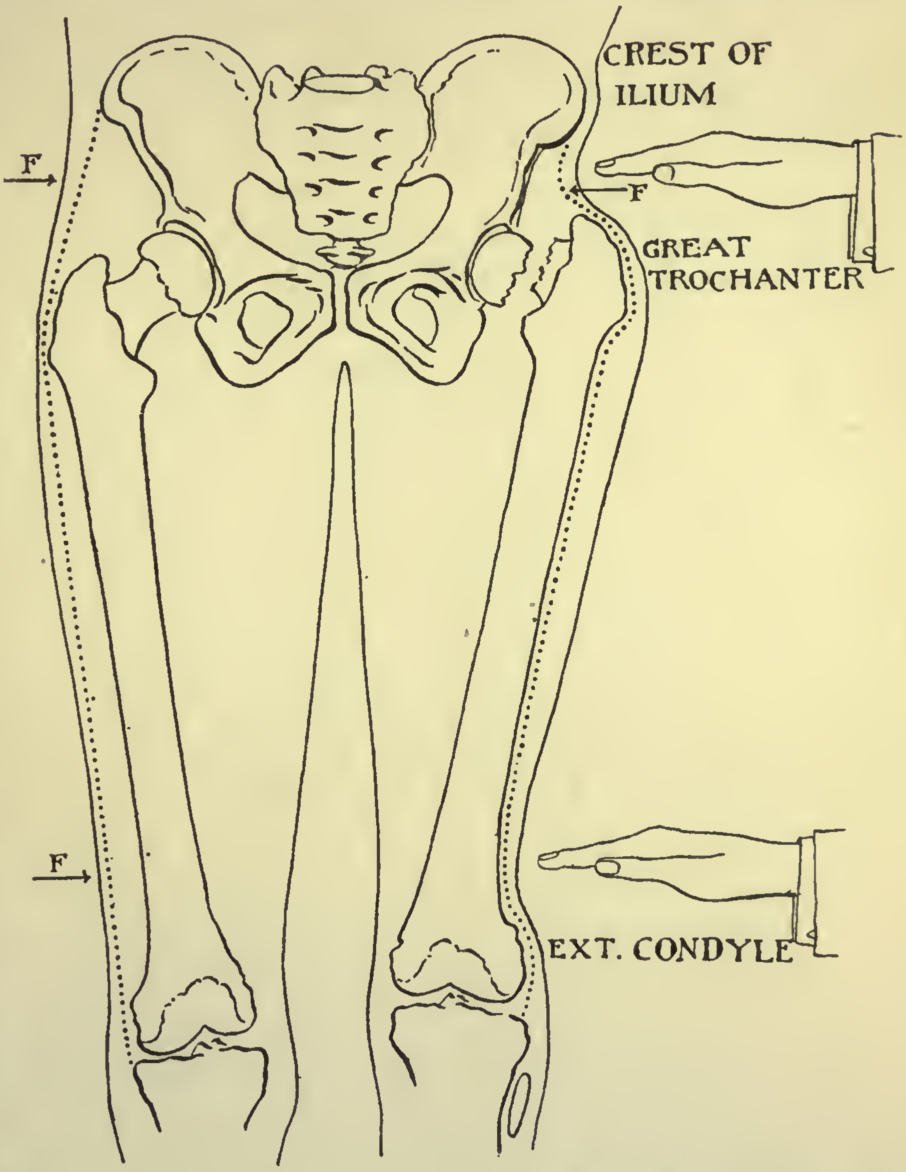

Fig. 5.—Diagram to show the manner in which the ilio-tibial band becomes relaxed in an intracapsular fracture of the femur. The diminished resistance of the ilio-tibial band on the affected side sometimes affords important corroborative evidence of the nature of the injury in doubtful cases. (From Allis's Gross Prize Essay.)

in consequence of the loss of resistance which is normally offered through the neck of the femur. Pain of a starting character is referred to the insertion of the psoas, and may extend down the thigh to the knee; it is increased by adduction and flexion of the hip. There is usually much loss of power over the limb, but when the reflected portion of the capsule is untorn the patient may be able to stand upon the injured limb. The experiment, however, is a dangerous one, and should never be tried, because so long as the two fragments are held in apposition there is some chance of obtaining bony union. Impaction rarely takes place, but when it occurs the neck is driven into the head of the femur. The prognosis is most unsatisfactory, both as regards the life of the patient and the use of the limb. Old persons with feeble circulations and atrophied lung tissues are liable to suffer from serious hypostatic congestion when they are suddenly compelled to lie flat upon their backs in bed. The temperature rises, there is some bronchitis, a little wandering of the mind, and in a few days, unless care be taken, and often when every care is taken, the patient dies: in other cases bedsores are a fertile source of trouble. In old people non-union is the rule in cases of intracapsular fracture. The non-union is either complete or fibrous, but bony union occurs sufficiently often to cause the surgeon to make every attempt to secure it unless the constitutional symptoms are so serious as to render it necessary to save life at the expense of the utility of the limb.

The treatment consists in applying an extension (vol. iv. p. 35) with a light weight of 2-3 lbs. to steady the limb and prevent the starting pains. A sandbag is placed on either side of the injured limb, and the patient is kept in bed for a month. A Thomas splint is then fitted and the patient is allowed to go about on crutches. If bedsores or any symptoms of bronchitis appear, the patient should be put into a Thomas splint at once and allowed to get up.

The measurements required by the instrument maker for the splint are as follows: the circumference of the chest at the line of the nipples; the circumference of the thigh two inches below the fold of the buttock; the circumference of the lower third of the leg about three inches above the malleoli. The length from the line of the nipples to two inches below the fold of the buttock; the length from the line of the nipple to the lower third of the leg about three inches above the malleoli. It must be stated for which side of the body the splint is needed, and if crutches are required, the distance from the axilla to the sole of the foot on the sound side must also be noted. A boot for the sound limb must be sent that a patten may be fixed to it. The price of a single Thomas splint varies from 6s. 6d. to about a guinea and a half.

Fractures of the neck of the femur occur in younger persons than the majority of intracapsular fractures. They are often known as extracapsular fractures, but incorrectly, because the capsule of the hip is attached in front to the intertrochanteric line of the femur, and the line of fracture is therefore only outside the femur posteriorly. These fractures are the result of direct and serious injury, such as falls upon the outer side of the great trochanter. The back of the neck of the femur yields first, and is often comminuted, the rest of the neck then breaks, and the proximal end of the bone is driven into the great trochanter. If the force is expanded the fracture remains impacted, but if it continue the bone is further split, and the trochanters are either detached or the great trochanter is so much comminuted as to set free the impacted neck of the femur. The fracture is not found more often in women than in men, as is the case in intracapsular fractures, and there is usually great pain made worse by pressure over the great trochanter. The limb is generally powerless, but the patient has control over it more frequently than is the case after an intracapsular fracture, and when the impaction is complete a few steps may even be taken with the injured limb.

The signs consist in a shortening, which, though slight at first, may suddenly become considerable owing to the separation of the impacted fragments. The great trochanter is increased in size directly after the accident; it lies nearer to the anterior superior spine of the ilium, and the hollow of the groin becomes filled up. The limb is generally everted owing to the posterior part of the neck of the femur being less resistant than the anterior portion, but a few cases of inversion have been observed. The ecchymosis and bruising are considerable.

The prognosis is good because firm bony union occurs in a comparatively short space of time. The impaction should be allowed to remain in people over middle age, who will then walk with a limp, but in young and vigorous adults the two fragments should be disengaged, in the hope that the union may take place without any shortening.

The treatment consists in extending and supporting the limb by means of a Hodgen splint, bent at the knee to an angle of 130°. The leg is first washed, dried, dusted with zinc oxide, and shaved if necessary. An extension apparatus of moleskin strapping with a wooden stirrup is secured to the leg and lower part of the thigh in the manner described (vol. iv. p. 35). Strips of house flannel are cut and arranged beneath the limb, from the great trochanter to the ankle, each strip overlapping the one above it, and each being three inches broad, and rather more in length than the circumference of the limb at the part it is to overlap. The splint is then placed over the limb so that the upper cross bar lies an inch below Poupart's ligament, and the lower cross bar is over the dorsum of the foot on a level with the instep, whilst the outer bar is as high as the anterior superior spine of the ilium, and the inner bar is opposite the tendon of the adductor longus. The strips of flannel are then pinned to the sides of the splint in order, and from above downwards, until the thigh and leg lie in a trough. A stout piece of blind cord is then attached to the stirrup of the extension, and to the end of the splint which projects at least five inches beyond the foot. The limb is slung by means of a pulley and cord attached to the splint by two pairs of hooks soldered to the sides, and the cord with a weight at the end runs over a pulley in a standard placed at the foot of the bed (Fig. 4). The position of the limb required to bring the two fragments into good apposition can only be determined by experiment, but the limb, even to its extreme upper limit, should lie free of the bed, and at the lower end the splint should be at least ten inches above it. The piece of cord attaching the stirrup to the end of the splint is a valuable test of the efficiency of the apparatus; so long as it remains taut good extension is being exercised upon the limb through the agency of the splint, but if it become slack the appliance must be re-adjusted. When a Hodgen splint is not available a long outside splint with a weight and extension may be applied for six weeks, and the patient may then be allowed to go about on crutches and in a Thomas splint for a further period of three to six weeks, the actual time depending upon his age and weight.

Fracture of the great trochanter is a rare form of fracture resulting from direct injury to the hip. The symptoms are flexion, deformity, limitation of movement. The great trochanter is drawn upwards and lies above Nélaton's line. It appears in some cases as if an actual increase had taken place in the length of the limb, and the accident is mistaken for a dislocation of the head of the femur, especially when the patient is only seen some time after the injury. Bony union generally occurs with the great trochanter in its new position, and the patient therefore limps after the accident. It is sometimes worth while to pin the great trochanter to the shaft, but in most cases the treatment consists in keeping the patient in bed with his limb between sand-bags or in a Thomas hip splint.

Coxa vara is a name given to a rickety incurvation of the neck of one or both femora which is seen more often in growing children than during the early years of life. Particular attention was first called to it in 1889 by E. Müller, who said that "in adolescence without apparent cause or following a slight injury, the patient begins to limp and to complain of fatigue and pain about the affected joint on exertion. Shortening of the limb is soon apparent and is caused by elevation of the trochanter above Nélaton's line. The limb is usually extended or flexed to a few degrees and somewhat rotated outwards. The motion of the joint is slightly diminished, particularly in abduction. There is no local tenderness on pressure." Coxa vara occurs in those who carry weights, do much walking, or are subject in other ways to prolonged fatigue. Its onset is marked by a peculiar stiffness of the hip referred to "growing pains," the stiffness being worse on rising after sitting for a time. It is relieved by complete rest. The shortening may be considerable, amounting to as much as 1½ inches. The prominence of the trochanters is especially well seen by flexing the thighs, and the displacement of the trochanter in relation to Nélaton's line is backwards as well as upwards. This is usually associated with external rotation of the limb, but in some cases there may be external rotation without any upward displacement. Tilting of the pelvis with consecutive scoliosis and secondary genu valgum or knock-knee may result from the deformity.

The diagnosis, prognosis, and *treatment* are discussed in article "Deformities," vol. ii. p. 452.

LITERATURE.—There is an interesting paper in connection with injuries of the hip by Mr. W. ARBUTHNOT LANE in the *Transactions of the Pathological Society of London*, vol. xxxvii. p. 387, 1886. It is entitled "The Causation and Pathology of the so-called Disease Rheumatoid Arthritis, and of Senile Changes." The quarto edition of Sir ASTLEY COOPER'S *Treatise on Dislocations and Fractures of the Joints*, issued in London in 1822, still remains unsurpassed for the clinical account of the dislocations of the hip as well as for the illustrations of this injury. The pathology of dislocations of the hip was first clearly described by H. J. BIGELOW (1818-1890), Professor of Surgery in Harvard University. His observations are published in *The Mechanism of Dislocation and Fracture of the Hip, with reduction of the Dislocations by the Flexion Method*, Philadelphia, 1869. Bigelow's work abolished the use of pulleys in the treatment of this form of injury, and substituted the method of reduction by manipulation. Professor Bigelow's pathology has been modified to a certain extent by Mr. HENRY MORRIS, *Trans. of the Medico-Chir. Soc.* vol. lx. p. 161, 1887; and more recently by Dr. OSCAR H. ALLIS, to whom the Samuel D. Gross prize was awarded in 1895, for his essay entitled *An Inquiry into the Difficulties encountered in the Reduction of Dislocations of the Hip*. The essay is published in 8vo, with numerous illustrations, Philadelphia, 1896. The treatment of unreduced dislocations of the hip is considered in the *Annals of Surgery*, vol. xx. 1894. The separations of the epiphyses in the neighbourhood of the hip are discussed in ample detail and with full bibliographies in Mr. JOHN POLAND'S excellent work *Traumatic Separation of the Epiphyses*, London, 1898. Fractures of the upper end of the femur are considered in Professor F. H. HAMILTON'S *Treatise on Fractures and Dislocations*, London, 1884, and by Mr. ROYAL WHITMAN in the *Annals of Surgery*, p. 673, June 1897. The Transactions of the British Orthopædic Society contain accounts of Coxa Vara.

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In individuals of average nutrition the constituent parts of the hip-joint are not available for direct examination. We are unable to detect the presence of fluid or the existence of synovial thickening so readily as we can in other joints, so that in the diagnosis of hip disease we are compelled to rely upon evidence which is indirect, *e.g.*, the presence of a limp in walking, an alteration in the attitude of the affected limb, restriction or loss of the normal movements of the joint, etc. The accurate interpretation of these phenomena necessitates considerable clinical experience, and no little care, on the part of the observer. He must be able to measure correctly the length of the lower limbs and of their different segments, to ascertain the position of the great trochanter in relation to Nélaton's line, to Bryant's triangle, and to determine the attitude of the lower limbs in relation to the pelvis, and of the pelvis in relation to the spinal column.

The hip-joint may be located as follows:—From the mid-point of a line drawn between the anterior superior iliac spine and the symphysis pubis a perpendicular line directed downwards will divide the joint into two equal halves. A horizontal line projected inwards from the upper edge of the trochanter will pass through the centre of the head of the femur; in children the trochanter is at a higher level, so that a similar line will touch the highest part of the head. The whole of the anterior and fully one-half of the posterior aspect of the neck of the femur is covered by synovial membrane, so that not only lesions of the epiphysis and epiphysial junction, but also those in the neck of the bone, are capable of direct extension to the synovial membrane and to the cavity of the joint.

While the capsular ligament of the hip is of extraordinary strength, there are certain weak points through which infective material may escape from the joint into the surrounding tissues; the most important of these is on the anterior aspect, where a bursa intervenes between the capsule and the ilio-psoas; the bursa communicates with the joint in one out of ten bodies; a second weak point exists immediately above the trochanter minor, where the tendon of the obturator externus passes above the insertion of the ilio-psoas; a bursa may intervene between these structures, and may communicate with the joint.

When the spongy bone constituting the floor of the acetabulum is the seat of infective disease the latter may extend within the pelvis, giving rise to a swelling which may be felt per rectum, and it may be also to complications involving the pelvic viscera.

Only these diseases of the hip are here described which present distinctive features. For other diseases the reader is referred to the general article on "Diseases of Joints."

TUBERCULOSIS. MORBUS COXÆ.

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Tuberculous disease of the hip, or "hip disease" as it is commonly called, without further qualification, is especially common in the class of patients who frequent hospitals. It is of great importance, inasmuch as it is a cause of prolonged invalidism or confinement to bed; it is attended with a considerable mortality (only second, so far as the skeleton is concerned, to disease of the spine and pelvis), and, especially if untreated, it may result in permanent deformity. The clinical study of hip disease is not unattended with difficulty, partly because the morbid changes are less open to inspection and palpation than is the case in other joints, and partly because of the complicated relationships of the lower extremity to the pelvis and spine in the various phases of the disease.

GENERAL FACTS AS TO AGE, SEX, RELATIVE FREQUENCY, ETC.—It is essentially a disease of early life; it rarely commences after puberty, and very rarely after maturity. In any case of hip disease in an adult one should be doubtful of its tuberculous origin, unless it is a relapse of antecedent disease.

Hip disease is slightly more common in the male than in the female.

The frequency of disease at the hip, in relation to that of other parts of the skeleton, varies with age; in children it comes second after the spine; in adolescents it comes third after the spine and knee.

In a certain number of cases hip disease is primary in the sense that it is the only discoverable tuberculous lesion; in others it follows upon tuberculosis of the lung, kidney, lymphatic glands, etc., and is thus to be regarded as definitely metastatic and less favourable.

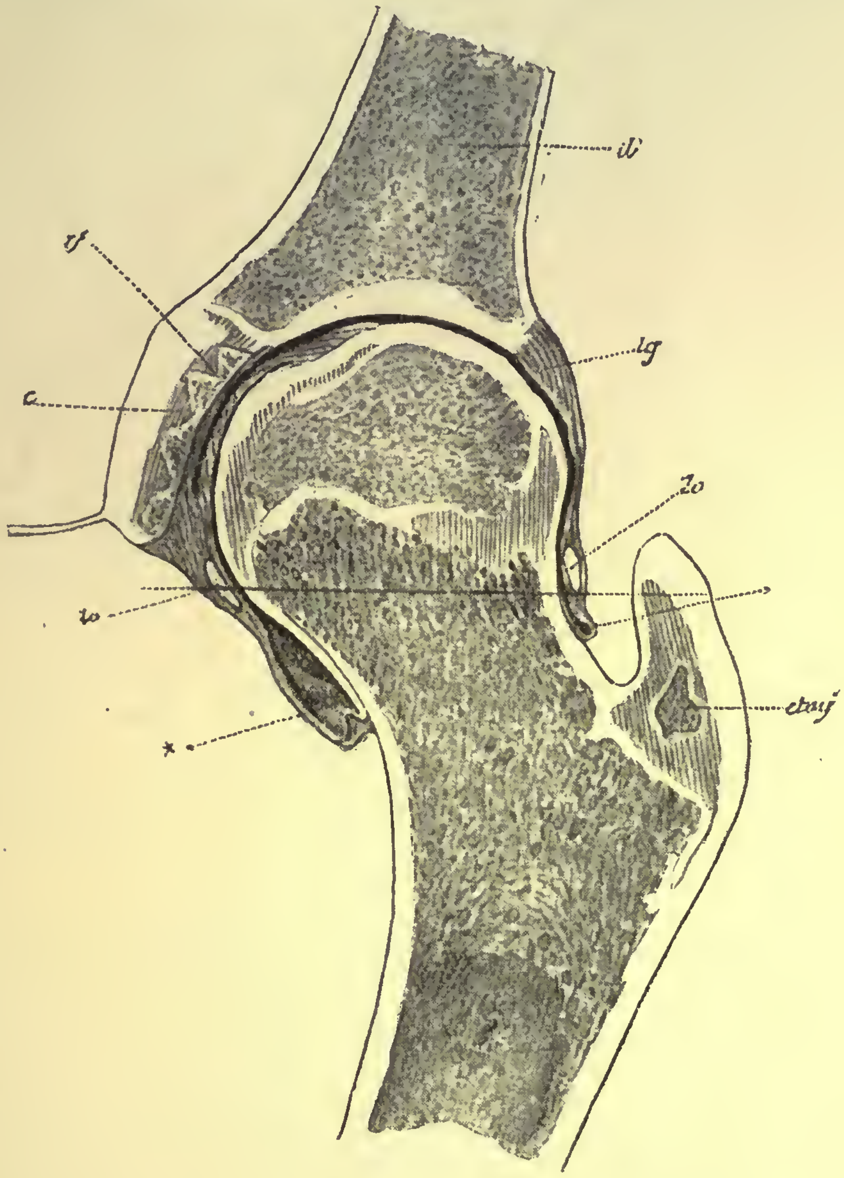
When it develops after an injury the patient is to be regarded as already tuberculous, and the injury merely plays the part of a localising factor.

PATHOLOGICAL ANATOMY.—This will only be considered in so far as it affords an explanation of certain of the clinical features, or affords guidance in diagnosis and treatment.

I. *The relative frequency of disease in the synovial membrane and in the bones* has an important bearing on prognosis; while recovery under conservative treatment may be reasonably anticipated in disease of synovial origin, operative measures will be required in the majority of cases in which there are lesions in the bones.

Observations based on specimens removed by operation or preserved in museums are misleading, as these are derived from the graver and more persistent forms of the disease. The general experience, however, is unanimous that bone lesions bulk more largely in the pathology of hip disease than is the case in other joints. The most recent estimate is that given by Krause based on 177 cases; the origin of the disease was osseous in 129, synovial in 26, and doubtful in 22. The proportion of osseous disease would appear to be even higher in the case of children; synovial disease *per se* is less infrequent in young adults, and in cases where other joints are affected simultaneously with the hip.

II. *Seat of the Bone Lesions.*—The acetabulum and upper end of the femur are affected in about equal proportion. The most frequent site *in the femur* is at or near the junction of the epiphysis and neck of the bone; in this situation the tuberculous material has only to erupt through the cortex of the bone in order to infect the synovial membrane or the entire joint. Gross lesions at the epiphyseal junction may cause detachment of the epiphysis, and the latter may subsequently become fixed in the acetabulum. Lesions in the epiphysis itself are on a smaller scale. Those at the base of the neck and in the trochanter, although capable of running an extra-articular course, are, if left to themselves, liable to extend to the joint; in exceptional cases they may do so by tunnelling the cervix and head of the bone. *In the acetabulum* the wall or the floor may be the original site of the disease; in either case the joint can scarcely escape infection, and the latter is usually more serious than when it arises in the femur.



III. *The nature of the bone lesions* has a considerable influence on the clinical features of hip disease; we may distinguish between the primary lesions, which are essentially tuberculous, and the secondary lesions, which are largely the result of pressure.

The tuberculous lesions present the same characters and wide variation as in other joints; it should be noted, however, that the sclerosed foci, which result in necrosis, are comparatively frequent, especially in the femur, where they are usually wedge-shaped, the base of the wedge abutting on the epiphysial disc or on the articular surface of the head. Riedel estimated that sequestra are present in 70 per cent of the cases subjected to operation. Disease in the floor of the acetabulum may result in perforation at one or at several points, and in the formation of an abscess within the pelvis. Gross alterations on the pelvic aspect of the acetabulum may be discovered on rectal examination.

The secondary changes from pressure involve an alteration in the size and shape of the bones subsequent to the destruction of the articular cartilage. While the disappearance of the surface bone may be the direct result of the tuberculous process, the more extensive alterations, at present under consideration, are the result of the absorption of bone, already softened by inflammation (rarefying ostitis), under the influence of pressure. In untreated cases, the bones, being immobilised in a definite position by muscular contraction, exercise a continuous and injurious pressure upon one another. *The head of the femur* undergoes absorption from above downwards; it loses its spherical shape, becomes flattened or truncated, or may disappear altogether; the neck of the bone in its turn may be absorbed. In the *acetabulum* the bone is absorbed in an upward and backward direction under the influence of the pressure exerted by the head of the femur; the socket is thereby enlarged and elongated towards the dorsum ilii; by active formation of new bone an attempt is made to maintain a buttress for the head of the femur; but the further progress of the disease may result in a further absorption of bone and a still greater enlargement of the socket; the lower and anterior part of the acetabulum, which is no longer in contact with the head of the femur, is filled up with granulation tissue, which may or may not be tuberculous. To this progressive enlargement and displacement of the socket Volkmann gave the suggestive name of "*wandering acetabulum.*" Its clinical importance is obvious, since the resulting displacement of the femur is the most important cause of actual shortening in advanced, and especially in untreated cases of hip disease; the displacement may be so considerable as to simulate a dorsal dislocation; the prominent posterior edge of the enlarged and displaced socket may be felt as a hard swelling above and behind the great trochanter.

Among the secondary results of tuberculous disease at the hip special mention must be made of *the occurrence of dislocation;* this may result from the alterations in the bones already described, or from destruction or softening of the capsular and round ligaments, or from a combination of these; the displacement of the head of the bone may be further assisted if the acetabulum has been largely filled up with granulation tissue. The direction of the displacement is nearly always upwards and backwards; the dislocation may be complete on to the dorsum ilii, or it may be incomplete; in the latter case the head rests on the upper and posterior edge of the acetabulum, and presents a deep groove corresponding to the line of contact.

The formation of abscess, which is such a frequent accompaniment of hip disease, usually proceeds from the joint itself, and the abscess usually communicates with the joint through an opening in the capsule. The abscesses within the pelvis usually communicate with the joint through the acetabulum. Abscesses may form in the vicinity of the joint from an extra-articular focus of tubercle at the base of the neck or in the great trochanter.

Clinical Features.—For convenience of description it is customary to divide these into stages, although never sharply defined from each other nor present in every case of the disease. There is usually a well-marked stage of onset characterised by a voluntary limp, by pain, and by restriction of the movements of the joint; a second stage with abduction of the limb and apparent lengthening, and a third stage with adduction and apparent or actual shortening. Of these stages, the first may be absent or may escape observation, the second is very typical of hip disease, but may also be absent, inasmuch as cases are met with of an acute type in which the attitude from the outset is one of adduction. The third stage is the one most often absent, either because the patient has recovered before reaching it, or because the adduction by which it is characterised has been prevented by treatment. The formation of abscess may take place at any stage in the progress of hip disease.

Initial Stage.—This probably corresponds either to disease which is still confined to the synovial membrane or to a lesion in the bone which has not yet opened freely into the cavity of the joint. The onset of hip disease is usually so insidious that the patient or his friends are rarely able to define the date on which it commenced. If injury is alleged as an exciting cause, some weeks usually elapse between the receipt of the injury and the onset of symptoms. In the case of a child it is usually brought for advice because it has begun to limp. The statement may be made that it has become pale, that it has ceased to take its food well, that its sleep has been disturbed, and that the pain and the limp, after coming and going for a time, have become more continuous and pronounced. On asking the child to walk, it may be observed that the affected limb is dragged in such a way as to avoid movement at the hip, and to substitute for this, movement of the pelvis; the child rests the weight of the trunk on the affected limb as little as possible, and inclines to rest on the balls of the toes rather than on the heel. There is usually some wasting of the muscles of the thigh and slight flattening of the buttock. Diminution or loss of the gluteal fold indicates a degree of flexion at the hip which might otherwise escape notice. Pain is complained of in the hip or shooting down to the inner side of the knee (through the obturator nerve); sometimes the pain is confined to the knee, and if the practitioner restrict his examination to this joint he may overlook the disease at the hip. At this early stage the attitude of the limb is not constant; it may be quite natural at one time and slightly flexed or abducted at another. Tenderness of the joint is of little diagnostic importance; it should be looked for either in front or behind the head of the bone; complaint of pain on driving the head against the acetabulum may occasionally assist in the recognition of hip disease, but the diagnostic value of the procedure has been overrated. Most stress is to be laid on *testing the functions of the joint*, and if it is carried out gently and without jerking there is no complaint of pain. The movements should be practised on the sound limb in the first instance in order to reassure the child that no violence is contemplated. The child should lie on its back, either on its mother's knee or on a table. On slowly flexing the thigh of the affected limb, it will be found that the range of flexion at the hip is soon exhausted, and that any further flexion takes place at the lumbo-sacral junction. The child is then made to lie on its face with the knees flexed in order to test the movements of rotation; the thigh is rotated outwards and inwards, and on comparing the two sides it will be found that rotation is restricted or abolished on the side affected, any apparent rotation taking place at the lumbo-sacral junction. These tests, as they may be called, reveal the presence of *rigidity* which results from the involuntary contraction of muscles; it is the most reliable sign of hip disease during the stage of onset; the procedure recommended has the decided advantage of being universally applicable, even in the case of very young children. If there should still exist any doubt as to the presence or absence of hip disease, the examination described should be repeated from week to week until one is able to arrive at a definite conclusion.

Second Stage.—The patient now presents more evident symptoms of hip disease, and usually exhibits the attitude of *abduction, eversion, and flexion*. This stage probably corresponds with commencing disease of the articular surfaces and progressive involvement of all the structures of the joint. The attitude described would appear to be the result of muscular action. The original view of Bonnet, that it is caused by distension of the capsule of the joint, is no longer accepted. The most reasonable *explanation of the various attitudes assumed in hip disease* is that given by König; if the patient walks without crutches, as he is usually able to do at this stage, the attitude of abduction, eversion, and slight flexion enables him to save the limb to the utmost extent; on the other hand, if he uses a crutch, as he is obliged to do at a more advanced stage of the disease, he no longer uses the limb for support, but draws it upwards and inwards into the position of adduction, inversion, and greater flexion; similarly, if he is confined to bed he lies on the sound side, and the affected limb sinks by gravity so as to lie over the normal one in the position of adduction, inversion, and flexion. König's explanation accords with the fact that in the exceptional cases which begin with adduction and inversion, we have usually to deal with a more severe type of the disease associated with grave osseous lesions, precisely those cases in which the patient is compelled to adopt the use of crutches or to lie up at an early period. Similarly the transition from the abducted to the adducted position is usually associated with such an aggravation of the symptoms that the patient is no longer able to walk without assistance.

The combination of abduction, eversion, and flexion, although characteristic of the stage of hip disease at present under consideration, is not constant; the proportion of the different elements not only varies in different cases, but one or other element may be wanting, *e.g.* abduction and eversion may occur without flexion.

The abnormal attitude is at first entirely maintained by the action of muscles, but where it is very prolonged or becomes permanent, the muscles, fasciæ, and ligaments undergo shortening, so that it is continued without muscular effort.

Compensation of Abduction, Eversion, and Flexion; apparent lengthening of the Limb.—On first looking at the patient the abnormal attitude may be largely concealed, because the patient usually restores the parallelism of the limbs, by lowering the pelvis on the affected side, and adducting the sound limb; this obliquity or tilting of the pelvis causes apparent lengthening on the diseased side; it is best demonstrated by drawing one straight line between the anterior iliac spines and another to meet it from the ensiform cartilage through the umbilicus; if the pelvis is in its normal or horizontal position, the two lines intersect one another at right angles; if it is tilted, the angles at the point of intersection are unequal. The flexion of the thigh may be largely compensated for by increasing the forward curve of the lumbar spine (lordosis), and by flexing the leg at the knee. To demonstrate the lordosis the patient should be laid on a flat table; when the thigh is flexed the lumbar spine is in contact with the table; when the thigh is extended the spine arches forwards so that one may insert the hand or the closed fist beneath it. In order to estimate the angle of flexion in any individual patient he should lie with the spine pressed flat on the table; the point is then determined, when, on attempting to extend the thigh, the spine begins to arch; the angle which the thigh forms with the surface of the table can be readily measured.

There may also be an attempt to compensate for the eversion of the limb by rotating the pelvis forwards on the affected side.

If *the functions of the joint* are tested by the methods already referred to, it will be found that there is more or less complete rigidity, and that both active and passive movements take place at the lumbo-sacral junction instead of at the hip. While the rigidity is usually absolute as regards rotation, it may sometimes be possible with care and gentleness to obtain a little increase of flexion; for diagnostic purposes most stress should therefore be laid on the presence or absence of the movement of rotation; for example, if a patient exhibits abduction and flexion, and it is possible to elicit rotation movements, the probability is that he is not suffering from disease in the joint, but from some affection in its neighbourhood.

Swelling, although rarely a prominent feature, should be looked for on the anterior aspect of the joint; when present it may fill up the fold of the groin, and may project forwards the femoral vessels; it is doughy and elastic like the white swelling of other joints, but may at any time liquefy and form a cold abscess; swelling about the trochanter and neck of the bone may be estimated by measuring with calipers and comparison with the sound side; the absence of swelling does not preclude the existence of grave lesions in the interior of the bone. Swelling on the pelvic aspect of the acetabulum may be discovered by rectal examination.

Third Stage.—The patient now presents unmistakable symptoms of hip disease characterised by adduction, inversion, increase of flexion, and apparent or real shortening of the limb. It probably corresponds with well-developed caries of the articular surfaces since pain is now a more prominent feature, and there are usually "startings at night." This stage is less often met with nowadays because the diagnosis and treatment of the disease in its earlier stages are better and more generally known. As far as *the attitude of the patient* is concerned, *the flexion* is usually so pronounced that it can no longer be concealed by lordosis, so that when the patient is recumbent, although the spine is arched forwards, the limb is still flexed both at the hip and at the knee; when the spine is flat on the table, the flexion of the thigh may amount to as much as a right angle. The *adduction* varies very much in degree; when moderate in amount it is compensated for by raising the pelvis on the affected side; this results in *apparent shortening* of the limb; it is of great importance to understand the cause of this shortening, since it can be prevented by treatment.

It is the result of an effort on the part of the patient to restore the normal parallelism of the limbs, the sound limb being abducted to the same extent as the affected limb is adducted. As a result of the obliquity of the pelvis, the patient when erect exhibits a scoliosis of the spine with the dorso-lumbar convexity to the sound side.

When the adduction is very pronounced the patient is unable to restore the normal parallelism of the limbs, and the knee on the affected side may cross the sound limb; there is a deep groove at the junction of the perineum and thigh, the pelvis may be tilted to such an extent that the space between the lower ribs and the iliac crest scarcely admits the fingers, and there is great prominence of the trochanter. In the slighter degree of adduction, which is more often met with, the toes of the affected limb rest on the dorsum of the sound foot, as in a traumatic dislocation on to the dorsum ilii.

The attitude described above is at first the result of muscular action, but when it is prolonged or permanent, the fasciæ, ligaments, tendons, and especially the adductor longus, become shortened, so that it may be impossible to correct it without division of the contracted soft parts.

If the case is left untreated further changes may take place as a result of the pressure of the carious articular surfaces against one another, viz. absorption of the head of the femur, progressive excavation and displacement of the acetabulum, so that the upper end of the femur is drawn gradually upwards and backwards within the socket. Examination will then reveal the existence of a variable amount of *actual shortening;* it will also be found that the trochanter is displaced above Nélaton's line, and that it is nearer the middle line of the body, while above and behind the trochanter there is a prominent hard swelling corresponding to the margin of the enlarged acetabulum.

At this stage there may be a combination of real and of apparent shortening which may amount to several inches. If, as very rarely happens, the original attitude of abduction has been maintained throughout, we may have a combination of apparent lengthening and of actual shortening.

In cases of long standing there may be an additional cause of shortening in the shape of retardation of the growth of all the bones of the limb. Shortening from dislocation will be referred to later.

During the third stage *the other signs and symptoms* become more pronounced: the patient becomes pale and thin; he is usually confined to bed; his sleep is disturbed by "startings" of the limb; the rigidity of the joint and the wasting of the muscles are very striking. Any elevation of the temperature is exceptional; it may rise slightly after a prolonged examination of the limb, or after a railway journey, or from abscess formation. Any marked elevation of the temperature, apart from septic complications, should make one suspect the development of tubercle elsewhere, or of general tuberculosis.

ABSCESS FORMATION IN HIP DISEASE.—The formation of abscess is not related to any particular stage of the disease; it may occur before there is any deformity, or it may be deferred until the disease has apparently recovered. The site of abscess and the direction in which it spreads is influenced by its seat of origin and by the anatomical arrangement of the parts concerned. The abscess may be solitary, or there may be several. They appear either in the thigh or in the pelvis, more commonly in the thigh.

The abscesses which form in the thigh usually originate from the cavity of the joint; less frequently they are periarticular; they may present in front of or behind the joint.

(1) *The anterior abscess* in exceptional cases ascends on the deep surface of the psoas muscle into the iliac fossa; it usually appears in the thigh, and comes to the surface on one or other side of the psoas; *the antero-external abscess* appears between the psoas and the great trochanter, a little below the anterior superior iliac spine; from the resistance offered by the fascia lata, it may gravitate down the thigh between the sartorius and tensor fasciæ femoris before perforating the fascia and erupting through the skin; *the antero-internal abscess* emerges at the inner border of the psoas, immediately above the trochanter minor, and bulges in the adductor region; it may perforate the pectineus muscle and point below Poupart's ligament to the inner side of the femoral vessels, or it may point anywhere from the perineum to the middle of the thigh. In connection with these anterior abscesses it occasionally happens that when they have opened and become septic the femoral vessels may be eroded, leading to serious and sometimes fatal hæmorrhage.

(2) *The posterior abscess* develops in the gluteal region, and may make its way to the surface through the gluteus maximus, more often it points at the lower border of this muscle in the region of the great trochanter, occasionally it gravitates down the thigh, or extends upwards towards the posterior iliac spine. Mention must also be made of *the extra-articular abscess in the region of the great trochanter*, which is often quite independent of the joint, and usually ruptures in the neighbourhood of the trochanter; also of *the abscess which originates in the lymphatic glands* along the femoral or external iliac artery, which may lead to the formation of tuberculous ulcers and occasionally to erosion of the femoral vessels.

The abscesses which form within the pelvis originate either in connection with the acetabulum or in relation to the psoas muscle where it passes in front of the joint. Those which are directly connected with disease of the acetabulum may remain localised to the lateral wall of the pelvis, or may spread backwards towards the hollow of the sacrum, or open into the bladder or rectum, or ascend into the iliac fossa and point above Poupart's ligament (usually to the outer side of the iliac vessels), or descend towards the ischio-rectal fossa and perineum and point in the vicinity of the anus, or pass through the great sacro-sciatic notch and point in the buttock. The abscess which develops in relation to the psoas muscle may be shaped like an hour-glass, one sac occupying the iliac fossa, the other filling up the adductor region of the thigh, the two sacs communicating with each other through a narrow neck beneath Poupart's ligament.

It has been well pointed out by Cheyne that *the key to the diagnosis of the point of origin of any pelvic abscess resulting from hip disease*, lies mainly in the examination per rectum; if a swelling is felt on the pelvic aspect of the acetabulum it is probable that the abscess has started there, and one may be able to recognise fluctuation between the iliac swelling and that felt per rectum. If there is no swelling in the region of the acetabulum, the probability is that the abscess has originated in the front of the joint, and has spread upwards into the iliac fossa along the psoas muscle.

The formation of abscess in relation to hip disease would be of comparatively little importance, were it not that by rupturing on the skin surface it results in the *septic complications* which are so disastrous, not only by aggravating the tuberculosis, but also by impairing the general health. The development and progress of abscess, so long as the skin is unbroken, is usually unattended with any fever, or complaint of pain, since unless the patient is carefully examined from time to time the existence of abscess may escape recognition. When the abscess bursts externally septic infection is almost inevitable; this may result in abundant purulent discharge, or where the external opening is insufficient for drainage, in repeated attacks of inflammation attended with fever, burrowing of the pus, and the formation of fresh sinuses, until the condition of the patient may merge into that of hectic fever or chronic septic poisoning, in which he loses ground from day to day, becomes the victim of waxy disease in the viscera, or dies of exhaustion, tuberculous meningitis, or general tuberculosis. We do mean to say that the presence of septic sinuses is incompatible with recovery, but they are a source of danger to which the patient should never be exposed if he comes under treatment with unbroken skin.

DISLOCATION.—True dislocation is a rare complication, and should never be allowed to take place in a patient who is under observation. It is more likely to occur during the stage of adduction with inversion. It is often ascribed to slight violence, but it is known to have taken place during sleep from the same spasmodic contractions of muscles that give rise to "startings" at night. In the more common dorsal dislocation, adduction and inversion are pronounced, the trochanter projects above and behind Nélaton's line, and the head of the bone may be felt on the dorsum ilii. In the upward or supracotyloid dislocation the head lies below the anterior superior iliac spine, flexion is little marked, eversion may replace inversion, the trochanter is farther from the middle line than on the sound side, the adduction is compensated for by tilting of the pelvis, so that there may be a combination of actual and apparent shortening.

It is a remarkable fact that when dislocation has occurred there may be less complaint of pain or of "startings" than before, and passive movements may be elicited which up till then were impossible.

BILATERAL HIP DISEASE. CROSSED-LEG DEFORMITY.—In the same way that any two or more joints may be simultaneously or successively the seat of tuberculous disease both hip-joints may become affected, each running its course independently of the other. The peculiarity of the bilateral affection is that the patient is necessarily confined to the recumbent posture until the disease has completely recovered, and that his capacity for standing and walking in the future may be seriously impaired, especially if the disease has cured with rigidity or ankylosis in both joints, and still more so if the joints have become fixed in an undesirable attitude; the length and the attitude of the limbs may not be the same; the most striking deformity is where both limbs are adducted so that they cross each other, this being known as the "scissor-leg" or "crossed-leg" deformity, in which the patient, if able to walk at all, does so by alternate forward movements from the knees. Where there is bilateral ankylosis the treatment consists in mobilising one joint by osteotomy or resection, and according to the result obtained, resting satisfied with the ankylosis on the other side, or correcting it by osteotomy or excision. If both hips have become rigid in the abducted position, one must be made movable by osteotomy or excision.

DIAGNOSIS OF HIP DISEASE.—The diagnosis is to be made not only from other affections of the joint itself, but also from morbid conditions in the vicinity of the hip, since in any one of these the patient may be brought for advice on account of pain in the region concerned and a limp in walking. Whenever possible the patient should be stripped; if able to walk, his gait should be observed; he is then examined in the recumbent position, and attention is directed to the comparative length of the limbs, to the attitude of the limbs and pelvis, and to the movements at the hip-joint, especially those of rotation. Whenever there is any doubt as to the diagnosis, both hips should be skiagraphed; the examination above described should be repeated at intervals of a week or ten days until a conclusion is reached. In children there are three non-febrile conditions attended with a limp and with shortening of the limb, which may be mistaken for hip disease, viz. *congenital dislocation, coxa vara, and infantile paralysis,* but in all of these the normal movements are less restricted than in disease of the joint, and the history of onset would be different. Cases of hip-joint disease which have recovered after the occurrence of dislocation may be difficult to recognise from the congenital variety in the absence of a trustworthy history, since the range of movement may be the same in both affections.

In sacro-iliac disease the pelvis may be tilted, and the limb apparently longer on the affected side, but the movements at the hip are retained.

In disease of the great trochanter the resemblance to morbus coxæ is very close indeed; there may be abduction, eversion, impairment of mobility, and swelling in the region of the trochanter, going on to abscess. The movements are less restricted than in disease of the joint, still the diagnosis may not be completed until the swelling about the trochanter is explored by operation, and this should not be delayed otherwise the disease may extend to the joint. The same remarks apply to *disease of the bursæ over the great trochanter,* especially of the bursa beneath the aponeurosis of the gluteus maximus.

In *lesions of the psoas muscle,* or of the *bursa underneath the psoas,* the limb may be flexed and everted, there may be lordosis, the patient may walk with a limp, and there may be no other signs of spinal disease; the diagnosis is made by testing the movements at the hip; in commencing psoas abscess, the movements are only restricted or absent in the direction opposed to the existing attitude, whereas in hip disease they are restricted or absent in all directions, especially abduction and rotation.

Appendicitis in children, when associated with flexion, has been mistaken for hip disease through omitting to test the movements of the joint in other directions.

Disease of the lymphatic glands in the groin may cause flexion and a limp in walking; it should be remembered that tuberculous lymphadenitis may be met with as a complication of hip disease, *i.e.* the two conditions may coexist.

Growth fever and other mild forms of osteomyelitis of the neck of the femur may simulate hip disease, but they are more likely to be attended with pyrexia, there is greater tenderness over the bone, the movements at the joint are less restricted, and there is more decided improvement after resting in bed.

Gummatous epiphysitis is a very rare affection met with in syphilitic children; it is attended with greater swelling and tenderness, and there is less voluntary power over the limb.

Sciatica has been mistaken for hip disease as a result of careless examination; in cases of sciatica flexion at the hip is only restricted when the leg is extended on the thigh.

New growths in the vicinity of the hip, and especially a commencing sarcoma of the upper end of the femur, may be very difficult to differentiate from hip disease, especially where the symptoms develop after a fall, when there is abduction or adduction, and where there is an ill-defined swelling about the trochanter. The movements at the joint are only restricted in certain directions in the case of sarcoma. Skiagraphs may give definite information. The recognition of egg-shell crackling or of pulsation is only possible in the advanced stages of sarcoma, and is therefore of little value.

The diagnosis from other diseases of the hip-joint is only difficult in exceptional cases.

In *traumatic effusion into the joint* the symptoms follow directly upon the injury, are more definite from the first, and rapidly improve under rest in bed without extension or splints.

Pyogenic affections of the hip, from staphylococcus osteomyelitis of the neck of the femur, from typhoid, from gonorrhœa, may be a source of difficulty especially when they are subacute and do not suppurate; there is usually, however, some pyrexia, there is greater tenderness, and pyogenic lesions may be discovered elsewhere in the body.

Charcot's disease and *arthritis deformans* usually occur under conditions which render any confusion with hip disease most unlikely; the author has, however, recently observed a case of arthritis deformans at the hip in a girl, aged 14, in which no other joints were affected, and in which the resemblance to hip disease was so close that in spite of repeated examination and of successful X-ray photographs, the nature of the case was not cleared up until the joint was explored by operation. In arthritis deformans there is less restriction of the movements at the joint, and if the patient is anæsthetised the rotation movements impart a sensation of roughness or crepitation.

In *hysterical affections of the hip*, while there may be the typical attitude and rigidity of tuberculosis, pain and tenderness are exaggerated, the pains are more diffuse, the tender points vary from time to time, and there is no swelling; when the attention of the patient is distracted it may be possible to elicit movement at the joint or to alter the attitude of the limb; the patient's sleep is rarely disturbed, and the condition may be of long duration without any signs of progress towards destruction of the articular surfaces.

PROGRESS AND TERMINATIONS. PROGNOSIS.—The prognosis in hip disease is more serious than in tuberculosis of other joints, excepting only those of the spine. It is materially influenced by the stage at which treatment is inaugurated, and by the care and perseverance with which it is carried out. While recovery may take place at any stage, and even in the most unlikely cases, it becomes less likely the longer it has been left untreated. In the initial stage recovery may be attended with a certain range of movement; the capacity of flexing the thigh to a right angle is a great advantage. When the disease has advanced to the stage of destruction of the articular surfaces, the joint will be rigid or ankylosed, and its use to the patient will vary with the attitude of the limb and with the amount of shortening. The average shortening amounts to 1½–2 inches, but it may attain to as much as 6 inches or more. The formation of abscess, especially within the pelvis, is a serious complication in untreated cases. The prognosis is most unfavourable where there are serious lesions of the bones (sequestra) and multiple infected sinuses, especially in adults who are the subjects of tuberculosis elsewhere. Intercurrent disease and unfavourable hygienic surroundings make recovery more difficult. The available statistics show that the mortality from hip disease varies from 10 per cent in cases of "dry" arthritis to 40 per cent in cases with abscess; the risks from abscess, however, have been much reduced in recent years.

The causes of death include meningeal, pulmonary, and general tuberculosis, septic complications, and waxy degeneration. In cases which terminate fatally the average duration of the disease is 1½–3 years. In cases which recover the duration varies indefinitely. Where there are bone lesions and septic sinuses the disease, which may have begun in childhood, may still be active after twenty years or more. It is a safe rule in practice to assume that even mild cases rarely recover in less than two years. The patient or his relatives should be informed of this, and also that they are not to expect recovery without some impairment of function, and that developments may occur which require more prolonged treatment or necessitate operative interference.

Treatment of Hip Disease.—As far as the local disease is concerned we know that a very large proportion of cases recover under conservative treatment, and that the functional results are very much better than those following operative interference, so that unless there are special indications to the contrary conservative measures are always adopted.

Conservative Treatment.—This consists in protecting the limb from injurious strain or pressure and in preventing deformity. Absolute rest is imperative, and the patient must be confined to bed until the pain and tenderness have entirely disappeared and the sleep is undisturbed.

Extension by means of the weight and pulley is of most service where there is pain, spasmodic contractions of muscles, rigidity of the joint, or a deformed attitude of the limb. Its object is to tire out the muscles and prevent the bones pressing injuriously against one another. The weight employed varies from 3-8 lbs. in children to 10-20 lbs. in adolescents and adults. It must be adjusted so as to meet the requirements of each case. If pain returns after having been relieved, the weight should be diminished or stopped for a time. If there is deformity the line of extension should be at first in the direction of the deformity, and should then be gradually altered as the attitude approaches the normal. Flexion yields most readily. In marked abduction extension should be applied to both limbs. In adduction it may be necessary to apply counter-extension by means of a perineal band. The attitude ultimately desired is one of almost complete extension with slight abduction. It is not necessary to apply the extension plaster to the limb as a whole; abnormal attitudes are more easily corrected if it is limited to the thigh.

The attainment of the desired results requires skill and close personal attention. Extension is too often employed in a routine fashion without regard to the special features of the case. There can be no doubt that the more general use of extension in the treatment of hip disease has been of great service in the prevention of deformity. It is capable of eliminating the common causes of shortening, viz. flexion, adduction, upward tilting of the pelvis, and displacement of the femur within the acetabulum. It cannot prevent the shortening from arrest of growth, but this can be compensated for by a moderate degree of abduction and tilting of the pelvis.

To further promote absolute rest in the attitude of extension and slight abduction additional means may be employed, especially in restless children: *a long splint* may be applied on the sound side, and a heavy *sand-bag* on the affected side; better still is a *double long splint and cross bar*. The long splint on the affected side is made with a hinge opposite the joint, so as to permit of abduction and of varying the amount of it. If there is any tendency to rotation it may be prevented by strips of strong plaster, wrapped round the thigh in a direction opposed to that of the threatened rotation, and fixed to the long splint (or sand-bag) with safety-pins.

Ambulatory Treatment.—When pain, tenderness, and muscular contractions have disappeared, and when any faulty attitude has been corrected (or over-corrected towards abduction), but not till then, the patient is in a condition to benefit from open-air treatment. The joint is still kept at rest, and the affected limb is not to touch the ground. In children who are unable to use crutches we rely upon a double Thomas splint, a Phelp's box, a Bonnet's wire cuirass, or plaster of Paris; by using any one of these the child is converted into a rigid object, capable of being carried about like a portmanteau from one room to another, or into the open air. Personally we have had best results from the double Thomas splint, like that intended for spinal disease, which extends from the occiput to the feet.

When the patient is able to use crutches the affected limb is prevented from touching the ground by fixing a high patten to the sole of the boot on the sound side. The hip-joint is kept rigid in the abducted position by a Thomas or Taylor splint, or by a case of plaster of Paris. The plaster is chiefly used in hospital out-patients who cannot be kept under continual supervision. It extends from the level of the navel to the foot on the affected limb and to the middle of the thigh on the sound limb. The plaster may be strengthened by broad strips of aluminium, and it should be renewed at intervals of from six weeks to three months.

The patient is re-examined from time to time, and the treatment is continued until some time after all symptoms have disappeared, and until that phase of hip disease which is productive of deformity is passed. The duration of treatment will vary from six months to two years. If at any time there is a return of active symptoms the patient must again lie up with extension to the limb. The patient or his relatives must be warned of the tendency to relapse.

Counter-irritation by means of the actual cautery has largely gone out of fashion, but where there is much pain in the persistent hip disease of adults it may give considerable relief. It is applied upon one or other aspect of the great trochanter. When there is *a deformed attitude which does not readily yield to extension*, such as may be met with in cases which do not come under treatment until it has existed for some time, there is considerable advantage in correcting the deformity at once rather than waiting until the disease is cured. This may be accomplished under anæsthesia in the majority of cases. If the tendons and fasciæ are so contracted as to prevent this they should be divided. The limb is placed in the attitude desired and maintained there by the methods already described. When there are other indications for operative interference (see later), the possibility of correcting a deformed attitude may be an additional incentive for recommending it.

Iodoform Injections into the Joint.—This procedure is recommended by Krause and others. The limb is placed in an attitude of slight adduction and inversion; the trocar and cannula, about 2 in. long, is inserted immediately above the trochanter major, and is pushed horizontally inwards at the level of the upper edge of the trochanter until it strikes against bone. This will either be the head or the upper part of the neck of the femur. The limb is then strongly adducted and the trocar is pushed on, always in contact with the femur, until it again strikes bone. This should be the socket. The trocar is withdrawn and the iodoform emulsion is injected through the cannula. The procedure should be repeated at intervals of two or three weeks.

Treatment of Abscess.—The early recognition and prompt treatment of abscess before it has enlarged, wandered, or threatened to rupture externally, has greatly reduced the suffering and mortality from hip disease. The methods of treatment are the same as are employed elsewhere, *i.e.* by means of iodoform injections or by open operation. If the latter is adopted, the opportunity may be taken of clearing out any extra-articular bone lesion at the same time. The wound should heal by first intention, or should be dealt with by the open method from the outset. If the abscess be extensive, or if it have relapsed, or if it be situated within the pelvis, the existence of serious bone disease should be suspected, and the question should be considered whether the joint itself should not be subjected to operative interference, either at the same time as the abscess or at a subsequent period.

Operative Treatment of the Disease in the Joint—Arthrectomy and Excision.—The results of conservative treatment are so superior to those obtained by the operations hitherto practised that we only have recourse to these when there is no prospect of spontaneous recovery. In other words, operation is indicated when the local conditions are such as prevent recovery, or when the general condition of the patient necessitates the removal of the disease. So far as the first of these indications is concerned, the difficulty lies in being able to recognise those cases in which the local conditions are incompatible with recovery at a time when it will be of practical value.

It must be remembered that complete excision of the hip is an operation of considerable severity, as evidenced by the existence of a considerable mortality from shock alone. The occurrence of tuberculous meningitis would appear to be as frequent in cases treated on conservative lines as in those subjected to operation.

Indications for Operative Interference.—1. When the disease from the outset presents unfavourable features, *e.g.* rapid progress, severe pain, early occurrence of adduction, or when it advances in spite of proper treatment; when pain and starting pains are not relieved by extension; or when, after a long period of quiescence, the disease becomes suddenly active and aggressive.

2. When the disease relapses after apparent cure, and if the recrudescence of the disease is attended with suppuration.

3. When, along with evidence of persistence of the disease, there is a deformed attitude which does not yield to extension; where there is dislocation of the head of the femur, or separation of its upper epiphysis.

4. When there is abscess within the pelvis, or other evidence of disease of the acetabulum.

5. When there are septic sinuses with symptoms of hectic and of waxy degeneration and persistent discharge in spite of proper drainage. These are most unfavourable cases whether an operation is performed or not. Operation may afford the only chance of recovery if it is not too long delayed, but it should not be attempted in hopeless cases.

6. In adults who show no signs of recovery within a year from the onset of the disease. This indication specially refers to "bread-winners," who desire above all things a definite promise as to when they will be able to return to work. Complete removal of the disease is an essential condition for any such promise. In patients over thirty natural recovery is so ex-

ceptional that it is unwise to count upon it. In children, on the other hand, there is no urgency for resuming active life; the prospects of spontaneous recovery are great, and an operation may interfere with the subsequent growth of the limb.

Nature of the Operative Interference.—From what has been said it is evident that we are often compelled to operate without knowing the condition of matters within and around the joint. We are, therefore, unable to decide beforehand the nature of the operation which is required. Our object should be to expose the disease wherever situated, and to remove it with the least possible damage to the normal structures. The more "open" the method of operation the better, since more reliable information is obtained by inspection than by palpation. These indications are best fulfilled by Kocher's method, which, by permitting of dislocation of the femur, affords free access to all parts of the joint. If it should be found that the disease in the head of the bone is limited to the surface or to isolated areas, it may be got rid of with the spoon or gouge, thus preserving the head, which is returned into the acetabulum at the close of the operation. This method, which may be described as an *arthrectomy*, should be practised wherever possible. If, on the other hand, there is a gross lesion in the head or neck of the bone, the neck is divided with a chain saw or chisel and the head is removed. The remaining disease having been got rid of, the raw surface of the neck is implanted in the socket, or is made to lie opposite to it. This operation is described as an *excision* of the joint.

In *the anterior operation* (associated with the names of Parker and Hüter) the head of the bone must be removed in order to give access to the acetabulum and posterior segment of the synovial membrane. As this excludes the possibility of arthrectomy, and affords an exposure of the joint inferior to that secured by Kocher's method, its application should be restricted to cases in which the lesion is believed to be confined to the anterior part of the joint.

Kocher's operation is performed as follows:—The patient is turned over on the sound side. An angular incision is made through the skin and fat with its centre at the anterior superior angle of the great trochanter. Its upper limb runs upwards and backwards in the line of the fibres of the gluteus maximus; its lower limb runs downwards and slightly backwards to the posterior margin of the base of the trochanter. The incision is deepened at the upper part of the wound so as to separate the fibres of the gluteus maximus. At the lower part it divides the tendinous insertion of the same muscle on the outer aspect of the trochanter, exposing the insertion of the gluteus medius. Blood-vessels are ligatured as they are divided. The edges of the divided maximus are held apart. A layer of fat is then divided, exposing the gluteus medius and minimus and pyriformis muscles. The wound is deepened in the interspace between the medius and minimus above and the pyriformis below. By drawing the pyriformis downwards, access is obtained to the posterior surface of the capsule and of the acetabulum. The capsule of the joint is divided along either the upper or the lower border of the pyriformis tendon. The insertions of the gluteus medius and minimus are detached from the trochanter from behind forwards, along with the periosteum and the cortex of the bone. The detachment is to be effected as far forwards as the anterior intertrochanteric line, and to allow of this the thigh should be flexed and rotated outwards. The ilio-femoral ligament is detached at the same time. The thigh is then flexed and rotated inwards to allow of the detachment of the insertions of the pyriformis, obturator internus, gemelli, and obturator externus from the inner and posterior surfaces of the trochanter and from the digital fossa. This separation should also include the periosteum and the superficial shell of the bone. In this way the muscles supplied by the superior gluteal nerve (the gluteus medius and minimus) are drawn forwards and upwards towards the tensor fasciæ femoris, which has the same nerve-supply, and which, along with the glutei, is of importance for the future abduction of the thigh, while the remaining muscles, which are mainly supplied by the inferior gluteal nerve, are displaced backwards. When the muscles are drawn apart the posterior surface of the head and neck of the femur and as much as is necessary of the great trochanter are exposed. Branches of the internal circumflex artery running transversely over the capsule at the neck of the femur are secured as they are divided. The diseased synovial membrane may be cut away with scissors from the posterior and upper aspects of the joint. The thigh is now strongly adducted, rotated in, and flexed, the ligamentum teres is divided by cutting on the head of the bone from behind and below; the head is then dislocated backwards, exposing the acetabulum and the anterior segment of the synovial membrane. The acetabulum is cleared of tuberculous tissue, special attention being directed to the removal of caseous or necrotic lesions. If the head of the femur is sound it may be replaced within the acetabulum; if it is diseased it must be removed. Iodoform is rubbed into all the surfaces exposed to the tuberculous infection. The divided ligaments, periosteum, and muscles are in turn sutured with catgut. A drainage-tube is brought out through a special opening behind the trochanter. The limb is fixed in an attitude of marked abduction, so that when the patient ultimately assumes the erect posture he will be obliged to depress the pelvis on the affected side in order to restore the parallelism of the limbs. The apparent lengthening thus induced will compensate, in part at any rate, for the actual shortening. The abducted position is secured by means of the double long splint already described. The drainage-tube is removed the day following the operation. The stitches are removed at the end of a fortnight. In a hospital patient a case of plaster of Paris is then applied for two months; thereafter he begins to walk with crutches.

As regards the result of the operation we are quite satisfied with a close fibrous ankylosis, for this ensures a strong and useful limb, and diminishes the chances of a relapse of the disease.

The anterior operation of Parker and Hüter is performed as follows:—The patient lies on his back. An incision is made through the skin and fat from a point immediately below and external to the anterior superior iliac spine downwards and slightly inwards for about three inches. The fascia lata is divided in the line of the wound. Search is then made for the interspace between the tensor fasciæ femoris and gluteus minimus externally, and the sartorius and rectus internally. The muscles being drawn apart, the capsule of the joint is exposed and freely divided; the head of the femur is then removed after dividing the neck of the bone by means of a broad chisel; the whole interior of the joint is now readily accessible to the finger, and is thoroughly cleared of all tuberculous material; special attention is directed to the neck of the bone and to the acetabulum. The operation is completed by stitching the edges of the divided capsule, the muscles, and fascia lata, and finally the skin. The limb is fixed in an attitude of marked abduction.

If in the course of either of the operations described an abscess is met with, it should be cleared out at once before opening up fresh layers of tissue. If there occurs a fresh outbreak of tuberculous disease in the scar of the wound, it is probably the result of infection of the tissues exposed during the operation, and it should be promptly dealt with by free excision of the parts involved before it ruptures externally. The formation of abscess at any subsequent period should be dealt with on the usual lines.

Amputation at the hip-joint for tuberculous disease has become one of the rarest operations in surgical practice. It may still be required in cases which have continued to progress in spite of excision, *e.g.* where there is disease of the pelvis or of the shaft of the femur, with septic sinuses, albuminuria, and hectic fever. In such cases the surgeon must be prepared not only to disarticulate at the hip-joint, but also to remove a portion of, or the entire innominate bone. Such extensive operations will usually be carried out by some form of racket incision, which will permit of the ligature of the main vessels as a preliminary measure.

The Correction of Deformity resulting from Antecedent Disease of the Hip.—The deformities concerned have been already described as accompaniments of hip disease; from neglect or improper treatment they may have been allowed to persist, while the joint disease has recovered. They are associated either with fibrous ankylosis of the joint, or with contracture of the soft parts, or a combination of these conditions. Osseous ankylosis is rare. The contracture of the soft parts specially involves the tendons, fasciæ, and ligaments on the anterior and inner aspects of the joint, and is usually present to such a degree, that even if the joint were rendered mobile it would prevent correction of the deformity. It should be borne in mind that the difficulties of correction are so considerable, that it should not be attempted unless the deformity is a serious hindrance to locomotion or a bar to remunerative occupation.

The deformity most often complained of is represented by a combination of shortening, flexion, and adduction.

If flexion is the prominent feature it may be corrected as follows:—The osteotomy is performed through the neck of the femur if it exists (Adam's operation), or through the trochanter if the neck of the bone has disappeared; the contracted soft parts are divided, either by subcutaneous or open tenotomy; the limb is then extended and abducted as described in excision of the joint. In patients whose occupations entail the sitting posture it may not be advisable to correct flexion.

If adduction and inversion are the prominent features the choice lies between a transverse osteotomy below the lesser trochanter (Gant's operation), an oblique osteotomy (from above downwards and inwards) through the great trochanter, or a resection of the joint according to the special features of the case. The adductor muscles, fascia lata, and other contracted soft parts are divided by subcutaneous or open tenotomy. The limb is then forcibly abducted and extended; the extension is maintained by the weight (10 to 20 lbs.) and pulley, the abduction by the double long splint already described. In cases where the deformity is bilateral special efforts should be made to obtain a movable joint, at any rate on one side.

SYPHILITIC DISEASE IN THE REGION OF THE HIP-JOINT is scarcely met except in the form of epiphysitis of the upper end of the femur in infants who are the victims of inherited syphilis; it is attended with swelling and tenderness over the neck of the bone, and diminution of the voluntary movements of the limb (pseudo-paralysis); while passive movements may be restricted, this is rarely so pronounced as in tuberculosis.

PYOGENIC DISEASES

These include a number of diseased conditions resulting from infection of the joint with the common pyogenic organisms, or with special organisms, *e.g.* the gonococcus, the typhoid bacillus. While the organisms usually gain access to the tissues of the joint through the blood-stream, a direct infection is occasionally observed from suppuration in the femoral lymphatic glands, or in the bursa under the ilio-psoas. The pyogenic diseases are chiefly met with in childhood and youth.

The clinical features vary with the gravity of the infection; they may be much less striking than is generally supposed. They may assume the form of an acute serous synovitis or hydrops, which recovers, or persists, or relapses after apparent cure. The features are often remarkably latent, especially when the hip affection occurs as a complication of a general illness such as scarlet fever, partly because the patient is confined to bed from the first onset. The hip lesion has been known to escape notice altogether, until at a later period the head of the femur was found lying on the dorsum ilii, or the joint is found to be ankylosed. In the "acute arthritis of infants," resulting from staphylococcus osteomyelitis of the femur or acetabulum, the clinical features may also be comparatively mild, but as a rule they assume a type in which the suppurative element predominates. There is general illness ushered in by a rigor and attended with an irregular remittent temperature; the local features are those of an acute arthritis; there is great suffering and frequently danger to life; the joint is held rigid, there is agony on the least attempt at movement, there are startings at night, the muscles waste rapidly; the limb may retain its normal attitude, more often it becomes flexed and abducted, or adducted; a swelling forms, usually in front of the joint at the upper part of Scarpa's triangle, pushing forwards the femoral artery; the swelling may be obscured by œdema; if untreated the pus escapes from the joint and either gravitates down the limb or, as is more often observed, it bursts through the skin over the joint and discharges by one or more sinuses; the joint becomes disorganised, the head of the bone may be displaced on to the dorsum ilii, or the upper femoral epiphysis may be detached; in the latter case the deformity resembles that which accompanies fracture of the neck of the femur.

As a complication of other diseases, pyogenic affections of the hip-joint are usually met with in relation to pyæmia and septicæmia (including puerperal fever), scarlet fever, typhoid fever, measles, acute rheumatism, and gonorrhœa.

In gonorrhœa the hip-joint is not uncommonly affected; the clinical features may be those of a serous synovitis with pain, tenderness and fever; the amount of fluid may be so considerable as to cause swelling in front of the joint. The more serious form of the disease is sometimes attended with great suffering and marked flexion, and may result in disorganisation of the joint and ankylosis. Suppuration is extremely rare. It is probable that some of the hip affections met with after childbirth are of gonorrhœal origin.

Progress and Terminations.—In the milder forms of pyogenic disease of the hip, recovery may take place with more or less complete restoration of function; in isolated cases the disturbance of growth at the neck of the femur may result in curvature (coxa vara). In more serious forms there may be persistent sinuses from disease (necrosis) in the femur or acetabulum, there may be fibrous or osseous ankylosis of the joint, permanent deformity from contracture of the soft parts, from separation of the epiphysis, from dislocation, and in children there may be impairment of the growth of the limb. In the gravest forms, especially where several joints or bones are involved, death may take place from general infection or toxine poisoning.

The occurrence of dislocation has been specially observed in relation to typhoid and other fevers, usually from some slight movement or push, when the limb is flexed and adducted; the dislocation may be the first indication of the existence of the joint lesion; the antecedent condition may be one of serous or purulent synovitis; destruction of bone, either of the head of the femur or of the rim of the acetabulum, is exceptional. If the child has been allowed to recover with dislocation on to the dorsum ilii, he is usually able to walk and run about, but with a limp which becomes more pronounced as he grows up; the features closely resemble those of congenital dislocation, and it may be impossible to distinguish between them unless an accurate history is obtainable. The movements are very free, except in the direction of abduction. The shortening may amount to 2 to 4 inches or more.

The treatment is conducted on general principles, and includes that of any disease of which the hip affection is a complication. The limb is extended by means of the weight and pulley, and kept rigid with the single or double long splint, or by means of sand-bags. If the swelling of the joint persist, and if there is evidence of suppuration, the joint should be explored by an anterior incision inclining downwards and inwards from the anterior superior iliac spine. In children it is remarkable how completely the joint may recover if the pus is promptly evacuated under rigid antiseptic precautions.

If there is *dislocation of the femur* it should be reduced by manipulation with or without preliminary extension; it has been successful in about one-half of the cases in which it has been attempted; with results which have been very satisfactory; preliminary tenotomy of the shortened tendons may be required in some cases. Where reduction by manipulation is impossible, the joint structures should be exposed by one or other of the methods employed in excising the joint; if the obstacle is found to be the filling up of the acetabulum by newly formed tissue, this must be removed; if the head of the bone has disappeared, the neck, or failing it, the trochanter should be fixed against the acetabulum, thereby diminishing the shortening at the expense of mobility. The author achieved this in one case, after dividing the shortened muscles, by driving a long steel pin through the trochanter into the acetabulum, and fixing the limb in the abducted position; the patient, a girl aged 15, was thereby enabled to bring both heels in contact with the ground and to walk very much better than she had done before. The treatment of gonorrhœal disease of the hip is the same as in other joints.

Acute Osteomyelitis in the Vicinity of the Hip-Joint.—This lesion may originate in the acetabulum or in the upper end of the femur, the latter being the more frequent; in many cases the disease runs its course without suppuration; extensive necrosis is exceptional; separation of the femoral epiphysis is nearly always attended with destructive changes in the joint. The disease usually affects children who are apparently healthy, it begins acutely with shivering, high fever, and severe pain, necessitating confinement to bed from the first onset. A doughy swelling forms in the region of the hip, usually in Scarpa's triangle. If suppuration takes place the features are those of acute abscess, which if left to itself results in the formation of sinuses. Rectal examination is of value in cases which originate in the acetabulum. Treatment is carried out on the same lines as in osteomyelitis elsewhere. The sequelæ which may be met with in neglected cases include contracture, deformity (usually extreme flexion with adduction), ankylosis, shortening of the limb, separation of the upper femoral epiphysis, spontaneous curving of the bone (coxa vara), fracture of the neck of the femur, and dislocation.

Death occurs in a considerable proportion of cases (about 15 per cent).

ACUTE AND CHRONIC RHEUMATISM OF THE HIP-JOINT

The joint lesions in acute rheumatism are described under that head. Chronic rheumatism is an ill-defined affection of joints; it is met with at ages varying from 12 to 50 years; it is usually polyarticular, and is chiefly remarkable for the amount of suffering to which it may give rise, and the great disturbance in the functions of the joint which may result from it. Its claims to be called rheumatic rest upon the facts that it has been observed to follow upon acute articular rheumatism, that it may show exacerbations or relapses attended with pyrexia, and that it is met with in patients who have a family history of acute rheumatism or of inflammations of serous membranes. It is probable, however, that some of the joint lesions described as rheumatic may be of gonorrhœal or other pyogenic origin. While it is regarded as distinct from arthritis deformans, it may merge into that disease, and ultimately present the alterations in the bones which are characteristic of it.

The clinical features of chronic rheumatism of the hip are similar to those observed in other joints; progressive stiffness and deformity are the outstanding features, associated with contracture of the soft parts and fibrous ankylosis of the joint; the patient very often becomes a helpless cripple, and has to spend the rest of his life in bed or on a bath chair.

The prognosis is unfavourable. Treatment is conducted on the same lines as in other joints. This subject is further described in the article "Rheumatism."

ARTHRITIS DEFORMANS—OSTEO-ARTHRITIS—MALUM COXÆ SENILE

This disease is comparatively common at the hip, either as a monarticular affection or simultaneously with other joints. While the etiology of the disease in general is obscure, in the hip it frequently develops after an injury.

The changes in the joint are characteristic of the "dry" form of the disease, and are chiefly met with in the cartilages and bones; the synovial changes which are so prominent in the knee are here of little clinical importance. The atrophy and wearing away of the articular surfaces is accompanied by the new formation of cartilage and bone around their margins; the head of the femur may acquire the shape of a helmet, or of a mushroom, or of a limpet shell; from absorption taking place in the neck of the femur, the head may come to be sessile at the base of the neck, and may occupy a level considerably below that of the great trochanter. The acetabulum "wanders" backwards and upwards as in tuberculous disease; it is usually deepened, its floor may project on the pelvic aspect, its margins may form a projecting collar which overhangs the neck of the femur, or grasps the latter so that even in the macerated condition the head is imprisoned in the socket, and the joint is locked. The eburnation of the articular surfaces resembles that seen in other joints. The degenerative changes may extend to the upper part of the shaft of the femur, and result in curving of the shaft and neck, suggesting a resemblance to a point of interrogation, and marked depression of the head of the bone below the level of the great trochanter.

The changes described are necessarily associated with great restriction of the movements of the joint and with striking deformity; the deformity consists in shortening of the limb, usually with eversion, sometimes with flexion and adduction; the trochanter is nearly always displaced upwards and backwards.

The clinical features are those of arthritis deformans in general, and are usually so characteristic that there is little difficulty in the diagnosis. The prominence of sciatic pain in some cases (from the nerve being involved as it passes over the joint) may lead to the disease being regarded as sciatica; the restriction of the movements of abduction and adduction, the presence of cracking and of grating of the articular surfaces, and the aggravation of the pain and stiffness after resting the limb, are helpful in recognising arthritis deformans.

The greatest difficulty is met with in cases where the disease occurs in early life as a monarticular affection, for the resemblance to tuberculosis may be so close that the diagnosis may be impossible until the joint is explored by operation; even skiagraphs may fail to differentiate between them.

The treatment, in addition to that of arthritis deformans in general, consists in maintaining the normal movements of the joint, and in diminishing the pressure on the articular surfaces by the use of sticks or crutches. Shortening of the limb may be compensated for by heightening the sole of the boot. Forcible movements of the joint under an anæsthetic often do more harm than good. Excision of the joint has yielded very satisfactory results; it is indicated where the patient, while healthy otherwise, is neither too old nor too fat, and is unable to walk on account of pain and deformity.

NEURO-ARTHROPATHIES

Charcot's disease is met with in individuals, usually men over 30, who are suffering from incipient or well-developed tabes dorsalis. One or both hip-joints may be affected. Sometimes the development of a fluctuating swelling in the upper part of Scarpa's triangle may indicate the presence of hydrops; in many of the recorded cases, however, attention has been first directed to the disease by the occurrence of dislocation from slight violence. The absence of pain and tenderness is always characteristic. When dislocation has occurred the limb is short, the upper end of the femur is freely movable on the dorsum ilii; the rotation arc of the great trochanter may be much reduced as a result of the disappearance of the head of the bone; there may be considerable formation of new bone, giving rise to large tumour-like masses in relation to the capsular ligament and to the muscles surrounding the joint.

When both hips are dislocated the attitude and gait are similar to those observed in bilateral congenital dislocation.

The treatment consists in protecting and supporting the joint; when only one joint is affected advantage may be derived from a Thomas or other splint, along with a patten and crutches, in bilateral cases, from the use of crutches alone.

HYSTERICAL JOINT.—The neuro-mimetic or hysterical joint affection is very rare at the hip; most importance attaches to the differential diagnosis, which has been already described under tuberculous disease.

LOOSE BODIES are chiefly met with in association with arthritis deformans and with Charcot's disease; they possess little or no clinical significance.

DISEASES OF THE BURSÆ IN THE REGION OF THE HIP.—The bursæ concerned are—(1) between the ilio-psoas muscle and the anterior aspect of the capsular ligament of the hip, which may communicate with the joint; (2) those in relation to the great trochanter, a superficial one between the skin and the tendon of the gluteus maximus, and a deeper one, often multilocular, between the gluteus maximus and the origin of the vastus externus; (3) over the ischial tuberosity; (4) beneath the attachment of the hamstring muscles to the ischial tuberosity; (5) between the obturator internus and the margin of the small sciatic foramen; this bursa may communicate with another between the tendon of the muscle and the hip-joint. The diseases met with in these bursæ include the so-called traumatic inflammations (trade-bursitis), and those resulting from tuberculosis, pyogenic infection, gonorrhœa, syphilis, gout, rheumatism, etc. (*Vide* article "Bursæ" in vol. ii.)

In disease of the bursa beneath the ilio-psoas the limb may be flexed, abducted, and rotated out as in tuberculous disease of the hip or of the psoas muscle; from the first of these the diagnosis is made by testing the movements at the joint, from psoas abscess by the swelling being confined to the region of the hip, viz. at the upper and inner part of Scarpa's triangle.

In disease of the bursa between the gluteus maximus and great trochanter the attitude of the limb may resemble that of hip disease; most difficulty will be found, however, in differentiating between tuberculous disease originating in the bursa and a cold abscess associated with disease in the trochanter itself; the diagnosis is rarely established until the swelling (or any sinus following on its rupture) is explored by operation.

Tumours in the region of the hip are so rare in comparison with other diseases that their existence usually remains unsuspected until their nature is revealed by the progress of the case. The *innocent tumours* include chondromata and osteomata arising from the trochanter or pelvis; the diagnosis is not difficult unless in exceptional cases; for example, *the bursa over an exostosis* may undergo rapid enlargement, and the patient may not be aware of the existence of the exostosis, and applies for advice concerning the bursa, which may be so large that it hides the tumour, and may be mistaken for a chronic abscess or other fluid swelling. The nature of the case may not be recognised until the swelling is incised.

Primary sarcoma may originate in the femur or pelvis; at an early stage the diagnosis from hip disease may be difficult or impossible; it has already been referred to under tuberculosis. The examination of the patient must include that of the rectum and the use of skiagraphs. In the later stages the nature of the disease is evidenced by distension of the cutaneous veins, œdema of the limb, pulsation of the tumour, egg-shell crackling, and the occurrence of spontaneous fracture.

Secondary cancer is by no means uncommon in the upper end of the femur following cancer in the breast, prostate, thyroid, intestine, or liver; it is not recognised as a rule until the occurrence of spontaneous fracture.

As far as *treatment* is concerned it is carried out on the usual lines in the case of the innocent tumours. In primary sarcoma it usually implies disarticulation at the hip by a racket incision, the handle of the racket being so placed as to permit of the ligature of the main vessels at an early stage of the operation. The soft parts are then divided in stages, well above the limits of the growth. There have been successful cases in which part or even the whole of the innominate bone has been removed along with the limb.

Paralysis of the Muscles at the Hip—Paralytic Contracture and Dislocation.—These are chiefly met with in children suffering from spinal or cerebral paralysis. At the first onset the limb is helpless and the joint is flail-like; if recovery takes place to such an extent that the child is able to walk, the attitude and gait may resemble that of congenital dislocation; where the affection is bilateral, the child has been known to walk with the hips acutely flexed so that the chest rests on the knees. The gait may be improved by some form of rigid apparatus which fixes the thigh to the trunk, or by securing an artificial ankylosis at the hip-joint (arthrodesis).

Cases are more often met with in which the child has been confined to bed (or at any rate is unable to walk), and contracture has taken place in the attitude of flexion, adduction, and internal rotation; dislocation on to the dorsum ilii may be superadded. It is of great importance to prevent such deformity by suitable splints and extension; when it has occurred, the question of correcting it will depend on the general condition of the patient and on the functions of the limb, apart from the attitude at the hip. The operative treatment is carried out on the principles recommended for similar deformities of other origin.

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Hodgkin's Disease. *See* LYMPHADENOMA.

Hooping Cough. *See* WHOOPING COUGH.

Hordeolum. *See* EYELIDS.

Horns. *See* SKIN, TUMOURS OF.

Hospitals.

HOSPITALS have been provided for the reception and treatment of the sick from earliest times. Hospitals supported by Government existed in India, Persia, and Arabia before the Christian era. It is declared that the sick were treated in the temple of Æsculapius as far back as 1124 B.C.

Within recent years a very great development has taken place in Scotland by the provision of hospitals for the treatment of infectious diseases, and also for ordinary medical and surgical diseases in cottage hospitals.

There are two distinct classes of hospitals: (1) Those for the treatment of infectious diseases, usually called isolation hospitals, and (2) those for the treatment of non-infectious diseases, such as ordinary medical and surgical diseases. In the first category the hospitals are usually provided under statutory powers by sanitary authorities, and the cost of equipment and upkeep fall upon the public rates.

There is no power by which sanitary authorities can provide hospitals for non-infectious diseases, as Section 66 of the Public Health Act states that any Local Authority may provide, furnish, and maintain hospitals for the use of the inhabitants of their district suffering from *infectious* disease. Space entirely forbids a lengthy description of the general principles to be observed in hospital construction, and it further demands that the description shall only refer to the smaller-sized hospitals suitable for villages and small towns and rural districts.

There will be no reference to maternity hospitals, hospitals connected with medical schools, asylums, workhouses, or with boarding schools; each of these would have required specialised reference to the special exigencies of the type.

There are certain general conditions to be considered in the construction of all houses, whether hospitals, schools, or ordinary dwelling-houses.

The first of these is the site.

Site.—Site has sometimes to be determined under conditions in which the hygienic element is not the most prominent. It should be dry and not malarious, and with an aspect which gives light and cheerfulness. The soil should be open and pervious, such as sand, gravel, and chalk. An underlying bed of clay is most undesirable. A clay soil is distinctly objectionable. Sites on granite, trap, limestone, and sandstone are usually healthy. Where the soil is damp, draining must be resorted to, and in all cases a damp-proof course of concrete or asphalt should cover the surface of the ground enclosed by the walls.

A site with a gentle slope at a moderate elevation facing the south, and protected from prevailing winds, is most desirable. A belt of trees, judiciously planted, may assist in moderating the intensity of winds.

The site should be removed from works which cause smoke and effluvial nuisances. Accessibility by main roads to the district the hospital is to provide for, to the medical attendant, to shops, telegraph and telephone communication, posting establishment, must all be considered. The area needed varies; in infectious diseases hospitals, about an acre for twenty-five to thirty persons is necessary, for non-infectious hospitals much less is required, but the amount will entirely depend on disposition and arrangement of wards, etc.

Construction.—This should be of such a nature as to ensure dryness of foundations, walls, and roof. The materials ordinarily used for walls are stone and brick, but concrete, corrugated iron, wood, are also used. Whatever material is used, the foundation should have a damp-proof course at a level of at least 3 inches above the ground.

Brick walls should be hollow, built with an air space of about 3 inches. The inner surface of the walls to be covered with some impervious material such as Kean's cement, "Adamant" plaster, or glazed bricks may be used. Angles at the junction of ceiling with walls, of walls with floor, of end with side walls, should be rounded off. Cornices, moulding, etc., should be avoided, in brief, all receptacles for dust should be avoided.

Floors should be made preferably of hard wood, oak or teak, or red pine, tongued and grooved, to be afterwards made impermeable by one or other of the carious compositions.

The floor space per bed for infectious diseases hospitals is usually 144 feet with a wall space of 12 feet. The width of ward 25 to 26 feet, giving a total air space of 2000 cubic feet per bed for infectious diseases hospitals. For small-pox cases, 3000 cubic feet are required.

Ventilation under the floors should be provided by opening in the walls at opposite sides. For the roof, slates laid on boarding and felt, provide the necessary means for securing dryness and warmth. Rhones and downspouts are necessary auxiliaries, and the roof water may be, with advantage, collected in tanks for washing purposes, at the same time simplifying the sewage purification problem.

Water Supply.—This should be wherever possible, by gravitation, and the water must be abundant and of quality above suspicion. Where sufficient "head" cannot be got, air-motors, rams, water-wheels, turbines, etc., may be necessary to pump up the water from some sufficient source. At least 50 gallons per head are required. Where the quality is not above suspicion, a Pasteur-Chamberland filter should be in operation.

Drainage, Sewage Disposal, and Purification.—In small country hospitals, earth-closets might be used, thus simplifying both the question of sewage removal and treatment. For isolation hospitals, the Local Government Board do not favour this system. They should only be used where pressing difficulties of sewage removal occur.

Whether an earth-closet or water-closet is used, it must be cut off from the ward by a passage with cross ventilation.

A wash-down closet with a three-gallon flush is to be preferred. Water-closet soil-pipes, sink and bath wastes, must be constructed on the most approved principles, but space does not permit of desirable details. Sewage, including water from baths and wash-house, must be purified before it is discharged into any stream.

The septic tank system; Ducat's or Dibdin's are the most likely methods now in use.

The methods of sewage treatment referred to depend on bacterial action. In the septic tank the sewage first undergoes anaerobic resolution, water, CO_2, CH_4, and H_3N being formed. The effluent is then run through coke beds, and nitrates are formed, where aerobic action occurs.

In Ducat's system the beds, made of coke breeze, are freely aerated by drain pipes passing into their interior. The sewage is distributed over the top by a sprinkler.

The beds in the latest development are covered over and warmed with hot-water pipes. The purification of sewage resulting is remarkable.

Where earth, peat, or saw-dust closets are used, some form of incinerator will be necessary. It will also be useful for burning poultices, old dressings, sweepings from wards, etc.

Ventilation, Warming, and Lighting.—For small hospitals ventilation is best secured by open fire-places and windows. There should be a window between every bed, and extending from 2 feet 6 inches from the floor to within one foot of the ceiling. One superficial foot of window for every 60 cubic feet of ward space is a fair allowance.

The windows should be double sashed and double glazed. Sometimes a "Hopper" light, hung on hinges, is provided in addition. Openings at the floor level with radiator ventilators, so that the incoming air is heated, are usually provided. Outlets may be further provided by Boyle, Buchan's, or other ridge ventilators. One inch of outlet for every 60 cubic feet of room is a usual ratio allowed.

Warming is provided best by open fire-places, supplemented as necessary by hot-water pipes either high or low (preferable) pressure, or by fire-clay stoves.

Such are the general conditions to be attended to in all hospitals, whether for infectious or non-infectious diseases, to secure abundant light, pure air at a proper temperature, dryness of the building, and prompt removal of offensive matters.

The essential parts of an isolation hospital are:—An administration block, containing nurse and matron's rooms, kitchen, scullery, pantries, larder, linen-store, storeroom, coal-store, nurses' and servants' bedrooms, bathrooms.

The extent of these will entirely depend on the size of the hospital, but cannot be very much curtailed for ordinary sizes.

The wards; and the best arrangement is to have single storey pavilions for each type of disease.

The sexes to be separated by an intervening nurse's duty-room, with a cooking range and hot-water arrangement.

By means of a closed window the nurse can supervise both wards. A bath on wheels can be kept in the passage or hall, and be used for both wards.

Steam disinfector, laundry and wash-house, ambulance shed, mortuary, coal and stick house, can form another block.

Caretaker's house and discharge rooms. The most suitable arrangement for all these is pavilions running north and south, administration block in front and in middle. Ambulance shed, laundry, etc., in rear. All separated by a 40-feet space.

Non-infectious diseases hospitals should have same conditions as to site, etc. already referred to. Accommodation will depend upon individual circumstances. It may be only a two or three-roomed cottage. One of the most recently established cottage hospitals is arranged thus.

The administration block is central, and the wards form wings in communication by passages with shutting-off doors.

On the ground floor of the administration block is a man's day-room and a woman's day-room, doctor and matron's room, and in the back, kitchen, scullery, and store, etc.

The two main wards are on a north-east line, and have six beds in each separate sexes. There are six private wards, four with one bed in each, and two with three. Above the central building are nurse's and matron's bedrooms, day-rooms, etc.

The floor space for the wards is 90 feet, and the cubic space 1350, and the general internal arrangements are as already described for isolation hospitals.

The operation room is on the ground level, and to the back of the building. The walls and floor are covered with glazed tiles. The operating table is of metal. Wash-hand basins and sink taps can all be wrought by the foot.

Glass tops resting on rubber are provided for the basins. Shelving is of glass on iron supports. A hose is provided for washing and flushing the floors and walls.

The lighting is from the roof. Disinfecting and sterilising rooms are provided for bedding, clothing, and for instruments. This hospital embodies the latest developments in hospital construction.

Rules for Visitors in Infectious Hospitals.—1. No person, unless an officer of the hospital or a member of the hospital committee, shall enter the hospital without a written order, signed by the Clerk or Medical Superintendent, except the Sanitary Inspector of any Local Authorities, who shall have right of entry only when bringing patients to the hospital.

2. Friends of patients will be admitted only when provided with a written order by the Medical Superintendent, except in cases where a fatal result is feared, when the Matron's sanction will be sufficient. Admission, under all circumstances, will be subject to the following conditions:—

(*a*) That not more than two visitors will be admitted at the same time.
(*b*) That each visit shall not exceed five minutes.
(*c*) That no fruit, food, or drink, or sweetmeats be given to any patient.
(*d*) That visitors shall not touch patients or their clothes, nor sit on their beds, nor go too near them.
(*e*) That they shall not smoke nor conduct themselves in any way disagreeable to the Matron or patients.

3. Visitors to small-pox cases must have been recently re-vaccinated, or have had the disease previously, and must cover themselves with an over-all dress to be provided for the purpose.

4. All visitors must obey the Matron's injunction in regard to length of visit, modes of preventing infection, etc.

LITERATURE.—1. *Burditt's Cottage Hospitals, and Hospitals and Asylums of the World.* 2. *Construction and Management of Hospitals*, by ROGER M'NEILL. 3. *Local Government Board Circular on Isolation Hospitals.* 4. *Isolation Hospitals*, by Sir R. THORNE THORNE.

Humerus. *See* FRACTURES, ELBOW AND SHOULDER JOINT.

Huntingdon's Chorea. *See* CHOREA.

Hydatid Disease.

HYDATIDS, TÆNIA ECHINOCOCCUS 50
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SYN.: *Echinococcus Disease.*

CORRESPONDING to each of the adult cestodes there is an immature form or larva, occurring usually in the tissues of an intermediate host, and developed from an ovum of the adult worm. The larva at a certain phase of its life-history exhibits the development of a "caudal vesicle," bladder, or cyst, and from this vesicle one or more heads, or scolices, may arise, which again develop, on gaining a suitable host, into the adult worm. In *Cysticercus cellulosæ*, the larva of *Tænia solium*, one head is formed from each bladder-wall. In *Cœnurus*, *e.g. C. cerebralis* (the cystic stage of *T. cœnurus*), which causes "gid" or "staggers" in sheep, many heads are budded out from the wall of the bladder, which is unilocular. The larva of *T. echinococcus* is also polycephalous, but the heads originate from the walls of special vesicles inside the primary bladder. The larvæ of *T. echinococcus* are termed echinococci, or hydatids, and their presence in the body constitutes the condition known as hydatid disease.

TÆNIA ECHINOCOCCUS is a cestode belonging to the family of *Tæniadæ*. It is 4-5 mm. in length, and consists of only three or four segments or proglottides in addition to the head or scolex, which has a transverse diameter of 0·3 mm. The head possesses four muscular suckers and a rostellum with two circlets of hooks, each circlet consisting of 14-25 hooks, which measure from 0·03 to 0·04 mm., those of the inner series being the larger (Leuckart). The hooks have stout root processes. The first segment is small, the second shows sexual organs, but the terminal segment, which is 0·6 mm. broad by 2 mm. long, and thus constitutes $\frac{1}{2}$-$\frac{1}{3}$ of the whole worm, is the only segment which is sexually mature. Like the proglottides of most other teniæ the terminal segment has a single marginal genital pore. The cirrhus (penis) and the cirrhus pouch are well developed, and the testicles number about sixty. The female organs consist of vagina, receptaculum seminis, oviduct leading to two ovaries, yolk gland, and uterus with short lateral ramifications. The uterus contains about five hundred ova, which are of about the same size as the ova of other cestodes (0·02-0·03 mm.), though the hard shell is somewhat thinner.

The *habitat* of *T. echinococcus* is the small intestine, where the white mature proglottides of hundreds or thousands of the worms may be seen protruding between the villi and showing lively movement. The dog is the chief host of this tænia, which is easily distinguished by its minute size from the other intestinal cestodes of the dog, and the tænia has also been found in the wolf (Cobbold), jackal, and a puma (*Felis concolor*). No other animal appears to naturally harbour the adult worm, and experiments have shown that *T. echinococcus* does not develop in the intestine of cats (Peiper), foxes, or rabbits (Leuckart), nor has it ever been found in the intestine of the human subject. The adult worm is developed from the scolices of the cystic larva, and dogs are naturally infected by eating the infected organs of an intermediate host. The metamorphosis of larval scolices into mature tæniæ occurs in 4-11 weeks, but as a rule is not complete till the seventh week (Leuckart).

HYDATIDS OR ECHINOCOCCI.—*Hosts.*—Mammals are almost without exception the only hosts. Sheep, oxen, swine, and man are the chief hosts, whilst the horse, kangaroo, goat, and other mammals are less often affected. Although the cystic worm in the human subject is termed *E. hominis*, and in the lower animals is called *E. veterinorum*, yet the specific identity of these two forms has been proved.

MODE OF INFECTION.—The mature proglottides or ova of *T. echinococcus* are deposited with the dog's fæces on the pasture, and the hard shell of the ovum to some extent protects the embryo inside. Herbivora are infected by feeding on the contaminated pasture. For the human subject, too, the dog is the sole source of infection. The licking of the human face and hands by dogs is often the means of infection, for the tænia may get on to the dog's tongue by migration, or in consequence of the dog vomiting. There may be ova or proglottides of the tænia on the dog's muzzle too, because of the dog snuffing at its own anus, or the ani or fæces of other dogs. Ova may also be deposited on the floor from the dog's muzzle or hindquarters, and toys, bits of bread, etc., may thus be contaminated. Again, the dog's fæces may be deposited in the neighbourhood of springs or pumps, and any proglottides or ova will tend to be preserved by the constant moisture, and will eventually reach the stomach in the drinking-water (Mosler). Many Australian writers assert that in their country infection is probably mainly due to drinking contaminated water. The eating either of uncooked vegetables, such as salad, or of berries and other fruits which grow near the ground, may also be a source of danger.

DEVELOPMENT.—If the ova of *T. echinococcus* enter the stomach their shells are digested, and the asexual six-hooked embryos are carried to the intestines, and bore their way (Leuckart) through the intestinal walls. The embryos thus reach venous or lymphatic channels, and are distributed throughout the body. As the embryos are mainly distributed by the portal system the liver is the most frequently affected organ. They may, however, be carried by the lymphatics to the mesenteric glands or peritoneum, and may gain the pulmonary or systemic vessels not only by traversing the liver, but also by the thoracic duct.

Having reached its destination in any part of the body the embryo loses its hooks, enlarges, becomes converted into a cyst, and excites a chronic inflammation of the surrounding tissues. The developmental changes were most thoroughly investigated by Naunyn and Leuckart. In the liver of a pig, four weeks after the ingestion of ova, Leuckart found small nodules each about 1 mm. in diameter. In the interior of each of these was the altered embryo, which is globular and resembles a mammalian egg, there being a thick, homogeneous, transparent outer zone enclosing coarsely granular material. Eight weeks after ingestion the larva had doubled in size, the granular material was clearer owing to partial liquefaction, and the fluid having collected towards the centre a cyst was formed, whilst the granular material situated more peripherally, just inside the homogeneous outer zone, had become differentiated into a definite layer, termed the parenchymatous or germinal layer, or endocyst. At this stage the endocyst consisted chiefly of granules and pale, drop-like cells, which sometimes showed stellate ramifications. When nineteen weeks old the cyst measured about 1 cm. in diameter, the homogeneous outer zone had attained a thickness of 0·2 mm., was distinctly laminated, and is called the external laminated layer, ectocyst, or cuticle. The ectocyst is formed by deposition in successive layers from the parenchymatous layer. The latter layer at this stage was not more than 0·12 mm. thick, yet could be differentiated into two layers—an inner consisting of small, clear droplets, and an outer layer of a more cellular nature —whilst there were highly refractile granules and calcareous corpuscles between the cells. External to the ectocyst is an adventitious fibrous tissue capsule formed by proliferation of the surrounding connective tissues.

Development of Brood-capsules.—When the cyst is about the size of a hazel-nut small elevations arise on the parenchymatous layer by cellular proliferation. The centre of each of these elevations becomes hollowed out, and the vesicle, or "brood-capsule," so formed, is bounded by two layers similar to those of the parent cyst; but in the brood-capsule the laminated layer lies internal to the parenchymatous layer. The older hydatids may contain thousands of brood-capsules, none of which, however, exceed 1·5-2 mm. in diameter.

Formation of Scolices.—Each scolex originates as a diverticulum from the wall of a brood-capsule, the cavity of the latter being continuous with the canal-like cavity of the diverticulum, which is, therefore, lined by a cuticle. At the distal end of this diverticulum, or rudimentary head, hooks and four suckers appear. The scolex is markedly contractile, and eventually becomes invaginated so as to project into the cavity of the brood-capsule; and the cuticle which formerly lined the canal is now the outer covering of the scolex, whilst the suckers and hooks are external too. The scolex, which is never more than 0·3 mm. in length, is now a solid cylindrical mass, attached posteriorly by a "stalk" to the wall of the brood-capsule; but the anterior or cephalic half of the scolex often becomes invaginated into the posterior part, so that the scolex has then a spherical shape. The hooks are smaller than those of the adult tænia, from which they are also distinguished by their small, slender root processes. Leuckart maintains that the bladder wall, brood-capsules, and scolices are all in direct structural continuity with one another, and that it is only as a result of exposure to deleterious influences, *e.g.* contact with water or death of the host, that the brood-capsules burst, when the scolices are either at once set free, or remain for a time attached to the shrivelled wall of the brood-capsule.

In many instances, and especially in the case of *E. veterinorum*, the larva undergoes no further developmental changes (acephalocyst), and growing but slowly it rarely exceeds an orange in size, and is either globular or irregular in outline, according to the resistance of the surrounding tissues. The ectocyst increases in thickness; and the adventitious capsule is often 5 mm. thick and very firm.

Formation of daughter-cysts, which have the same structure and function as the mother-cyst.—1. *E. exogenus* or *E. granulosus*, where daughter-cysts lie outside the mother-bladder. These daughter-cysts originate as buds in the deeper layers of the maternal ectocyst (Leuckart), the buds enlarge, push forward, and finally burst through the superficial layers of the maternal ectocyst. They form an external cuticle for themselves, their central portion becomes liquefied just as in the case of the parent cyst, and the daughter-cyst may finally be completely separated from the parent cyst by the fibrous capsule of the former. *E. exogenus* is most common in the domestic mammals, especially the pig; whilst in man it is rarer, and has been found mainly in the bones and on the peritoneum, though in the liver or other organs also.

2. *E. endogenus* or *E. hydatidosus*, in which daughter-cysts lie within the parent cyst. The daughter-cysts may be few in number and of globular form, or may number some hundreds or thousands, and then from mutual compression

exhibit irregular forms. They originate in retrograde metamorphosis of scolices and brood-capsules, which thereby assume the structure and functions of daughter-bladders. If they be formed from brood-capsules the superficial parenchyma of the latter disappears, the scolices lose their form, and ultimately become a layer which is evenly distributed over the *internal* surface of the cuticle, thus forming a new parenchymatous layer. Daughter-cysts may also be formed from folds of the maternal parenchyma, portions of which form a cuticle, and become hollowed out internally in the same way as exogenous cysts.

In either of these varieties granddaughter-cysts may arise from the daughter-cysts just as the latter do from the mother-cyst.

3. *E. multilocularis* is usually found in the human liver, and varies in size from that of a pea to that of a child's head. On section it is found to consist of numerous small spaces (0·1-4 mm. in diameter), enclosing clear granular substance, and separated from one another by a fibrous stroma, so that the appearance resembles that of a colloid carcinoma. The multilocular echinococcus has a tendency to degenerate at its centre and to become calcified. The line of separation between the echinococcus and the surrounding liver tissue is very irregular, and the liver is often bile-stained. This variety has also been found affecting the lungs, peritoneum, and brain, but is very rare in other situations. The spaces between the fibrous stroma, like the bladders of other echinococci, possess a laminated cuticle, but scolices are seldom developed. *E. multilocularis* probably originates, not in consequence of infection by many embryos, but from one embryo by repeated exogenous proliferation, a mode of development somewhat similar to that of the racemose forms of echinococci seen in the livers of cattle.

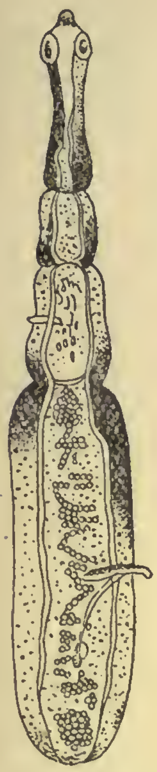

FIG 1.—Adult Tænia echinococcus (×12). Leuckart.

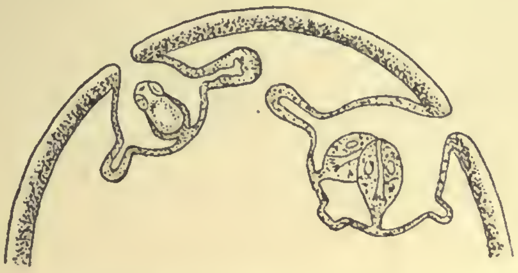

FIG. 2.—Diagrammatic representation of a proliferating echinococcus. Leuckart.

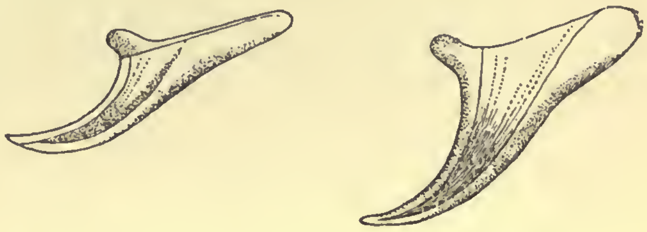

FIG. 3.—Hook of Echinococcus veterinorum (×600). Leuckart.

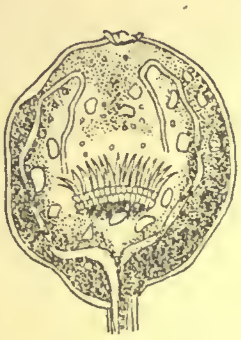

FIG. 4. — Echinococcus-head, with the anterior part of the head invaginated (× 90). Leuckart.

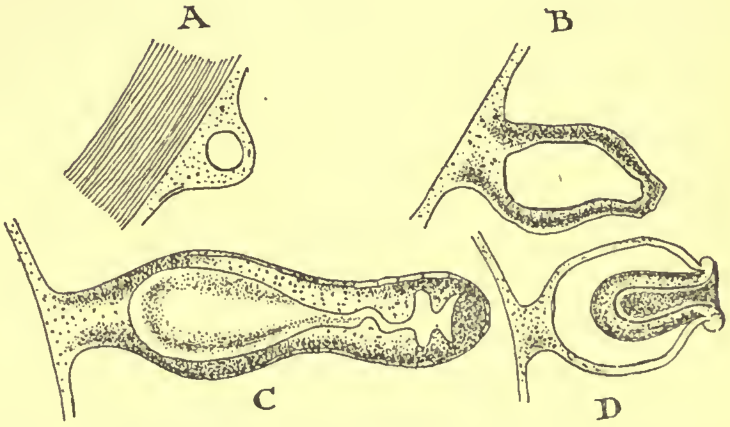

FIG. 5.—Development of brood-capsules (A) and of the appended heads; (B) first rudiment of the head; (C) further development; (D) invagination (× 90). Leuckart.

The laminated ectocyst is highly elastic, and when incised curls upon itself. It possesses considerable capacity of imbibition, and contains chitin and a small amount of hyalin (Hoppe-Seyler).

Hydatid fluid is colourless, transparent, of neutral alkaline or faintly acid reaction, and has a specific gravity varying usually from 1009 to 1015. It usually contains 97-98 per cent H_2O, 0·5-0·8 per cent NaCl, succinic acid and inosite. The fluid may contain other constituents owing to the absorptive power of the ectocyst; the fluid of a hydatid in the liver may contain grape sugar, leucin, tyrosin, cholesterin, cystin, and hæmatoidin, whereas in the kidney it may contain crystals of uric acid, oxalic acid, triple phosphates, and other earthy salts. The normal fluid never contains any coagulable albumin, which can as a rule, however, be detected if the cyst wall is inflamed or has been previously punctured. In the latter case the presence of albumin is possibly due to functional changes induced in the hydatid by the puncture.

Changes liable to occur in the Hydatid Cyst.—The echinococcus often dies spontaneously. Degenerative changes commence just outside the ectocyst; the fluid becomes opaque, coloured, albuminous, and reduced in amount; the scolices fall off, and the hyatid is represented by a pulpy débris which still contains the hooks and possibly fragments of ectocyst, and which, together with the capsule, may undergo calcareous infiltration. Partial ossification has been observed in rare cases. Death of the larva may also be caused by evacuation of the fluid it contains, or by suppuration commencing round the cyst and spreading to its interior. Rupture of the cyst is another possible occurrence, either as a result of trauma or of atrophy of surrounding structures from pressure.

Geographical Distribution.—Statistics from Iceland represent 1·2-3 per cent of the population as affected with hydatids. Hydatids do not occur in the Faröe Islands (Davidson). In German-speaking countries patients with this disease formed 0·03 per cent of a total 500,000 cases treated in hospital (Neisser), but the disease is comparatively rare in Southern and Central Germany, while more common in the north, especially in Mecklenburg and Pomerania. For example, in Rostock there is one case of hydatid disease for every 1056 inhabitants. The following figures illustrate the frequency of the disease in different parts of Germany (Peiper):—

In Kiel,	1872-1887,	hydatids were	found in	0·19	per cent	of total	autopsies.
In Munich,	1854-1887,	„	„	0·25	„	„	„
In Jena,	1866-1887,	„	„	0·84	„	„	„
In Greifswald,	1862-1894,	„	„	1·50	„	„	„
In Rostock,	1861-1883,	„	„	2·43	„	„	„

In Switzerland ordinary hydatids are rare occurring in 0·13 per cent of all autopsies. In Vienna (1892-1897) hydatids occurred in 0·02 per cent of hospital patients. In Dalmatia, however, the disease appears to be more common than in any other part of Europe. In Servia 0·02 per cent of hospital patients are affected. In Belgium and Denmark the disease is rare, only four cases are reported from Norway, in Italy and France hydatids are more often seen. In Great Britain hydatid disease is uncommon, but it is more frequent in England than in Scotland (Murchison). In *St. Thomas's Hospital Reports* 1894-1898 there are 19 cases; in *Middlesex Hospital Reports* 1893-1897 there are 15 cases; in *Westminster Hospital Reports* 1889-1898 only 10 cases; and in *Edinburgh Hospital Reports* 1891-1897, 11 cases are recorded. In South Africa, Egypt, China, and India, hydatid disease is rare. Osler could find records of only eighty-five cases in the United States and Canada up to July 1891. In the Argentine Republic the disease is common, but very rare in other parts of South America. The disease is common in some parts of Australia, especially in Victoria and South Australia. The average death-rate per 100,000 from this cause during the period 1882-1886 was as follows:—Victoria, 5·79; South Australia, 3·54; New South Wales, 2·05; Queensland, 1·29. In Tasmania hydatids are rather common, giving a death-rate of 2·83 per 100,000, but in New Zealand the corresponding mortality is only 0·66 per 100,000 (1882-88, Davidson).

Thus the disease is most frequent in Iceland, Australia, Dalmatia, and parts of Northern Germany.

Posselt has recently collected records of 215 cases of multilocular echinococcus. Their geographical distribution, which is very remarkable, is as follows:—1. Bavaria 56, Austria (the majority in the Tyrol) 30, Switzerland 27, Würtemberg 25, Baden 3, etc., in short 147 cases within a circumscribed area; 8 cases in districts adjacent to this area; 40 cases in the two Russian territories of Kasan and Moscow. 2. 20 cases which occurred irregularly in various countries. No case has occurred in Great Britain. There is as yet no definite proof that multilocular echinococcus is derived from a special variety of *T. echinococcus.*

The following factors favour the occurrence of hydatid disease:—

I. A high percentage of dogs infected with *T. echinococcus.* 28 per cent of dogs were said to be affected in Iceland, and 6 per cent in Copenhagen (Krabbe). The tænia is a common parasite in Mecklenburg (Madelung), and was found in 40 per cent of unregistered dogs in Australia (Thomas). In England "probably at least 1 per cent of our dogs harbour the mature tape-worm" (Cobbold), whilst in North America it is a rare parasite (Osler).

The actual frequency of the dog is of less importance. For example, the ratio of dogs to individuals is 1 : 11 in Iceland, 1 : 18 in Mecklenburg (Madelung), and the ratio of registered dogs to persons in Australia is 1 : 23, whilst the registered dogs constitute only a fractional portion of the total (Graham), yet the ratio is 1 : 27·8 in Pomerania, 1 : 22 in France, and 1 : 15 in Belgium.

II. Circumstances facilitating the transmission of the ova to the human subject:—

The disease occurs most frequently in those who are brought much in contact with dogs, *e.g.* Australian shepherds, and is more common in country parts than in towns. The disease is common too in Iceland, where during the long winters the dogs live in the same room with the people, many of whom are so careless that instead of washing their dishes they permit them to be licked clean by the dogs (Leuckart).

III. Many domestic mammals, and, still more important, the frequent occurrence of *E. veterinorum* in them.—For every hundred persons in

		Cattle.	Sheep.	Swine.	
Australia there were . . .	(1892)	300	3000	...	(Verco and Stirling)
Iceland „ „	(1883)	46	488	...	(Krabbe)
Mecklenburg-Schwerin there were	(1884)	47·3	164·4	39·5	(Madelung)

in Pomerania there were 168·4 sheep to every 100 persons, whereas for every 100 persons in

	Cattle.	Sheep.	Swine.	
Italy there were . . .	16·8	30·2	4·1	(Madelung)
France „ . . .	30·4	59·8	14·8	„
United Kingdom there were	28·2	86·4	8·2	„

and in these countries *E. hominis* is comparatively rare.

Again, the prevalence of hydatid disease in man is proportionate to the frequency of the disease in the domestic animals. Peiper has shown this to be the case in the various districts of Mecklenburg, *e.g.* in Greifswald, where hydatids are common in man, 64·58 per cent of cattle, 51·02 per cent of sheep, and 4·93 per cent of swine are affected. In other parts of Germany not only man but the domestic animals too are comparatively rarely affected, *e.g.* in Karlsruhe 2·90 per cent of cattle, 2·24 per cent of sheep, and 0·66 per cent of swine have hydatids. In Australia about 44 per cent, and in the Argentine Republic, 30 per cent of sheep are affected, and in Iceland the disease is common both in sheep and cattle.

The occurrence of *E. veterinorum* is favoured by a mild damp climate, in consequence of which the ova of *T. echinococcus* may lie for months on the pastures and still be capable of development. It is the animals kept at pasture during the summer which are most frequently infected.

IV. Circumstances facilitating the metamorphosis of the larval scolices into adult tæniæ.—The more frequent the occurrence of *E. veterinorum* in the animals slaughtered the greater are the risks of dogs becoming infected. For the diseased viscera are often carelessly disposed of, or even employed to feed the dogs. In the absence of proper slaughter-houses, as in some parts of Mecklenburg until comparatively recently, dogs are likely to acquire infection.

Hydatid disease in relation to the *occupation* of the individual.—It is most common in those who are much in contact with dogs, *e.g.* shepherds. The possible modes of infection are, however, very numerous, and the disease has been observed in joiners, tailors, and others, though very rarely in seafaring individuals.

Sex.—The relative frequency in the two sexes depends largely on their occupations and pursuits. In Iceland the disease is more common in women, as Finsen in 255 cases found 181 in the female sex. Neisser found 436 cases in females out of a total of 669; Davaine and Peiper hold that the sexes are equally affected; but in Australia hydatids are more common in men; the ratio being 100 : 77 (Verco and Stirling).

Age.—Hydatids occur at almost any age, but are rare in children. Neisser gives the following figures:—0 to 10th year, 4·8 per cent; 11th to 20th year, 13·2 per cent; 21st to 30th year, 30·8 per cent; 31st to 40th year, 24·6 per cent; 41st to 50th year, 15·2 per cent; 51st to 60th year, 6·2 per cent. Hence the disease is most commonly seen between the ages of twenty-one and forty, and is rare after sixty.

Anatomical Distribution.—In swine the liver is more often affected than the lungs (18 : 2·3), in sheep the liver is less often affected than the lungs (14 : 21), whilst in cattle these organs are about equally affected (7·5 : 8·2) (Peiper).

According to Neisser's statistics of 900 cases, the distribution in the human subject was as follows:—Liver 451, lung 67, spleen 28, pleura 17, heart or vessels 29, cranial cavity 68, spinal canal 13, kidney 80, pelvis 36, female genitals or mamma 44, bones 28, face orbit or mouth 21, other parts 18. As a rule only one organ is affected, and in 50 per cent of cases it is the liver.

In some cases, however, there are *multiple echinococci.* In animals these are of common occurrence, and of comparatively small size. In man the liver usually contains one echinococcus, whilst there are echinococci in other situations too. The peritoneum is one of the sites specially affected, for here a solitary echinococcus is rare, but several hundreds are not uncommon. The cause of multiple echinococci is variable. They are, however, usually referred to infection by numerous embryos at one time, to repeated infection, or lastly, and especially in the case of the peritoneum, to development from daughter-bladders, brood-capsules, or scolices which have escaped from the parent-cyst in consequence of tapping or rupture.

An illustrative case has been recently recorded by Subbotic. At the post-mortem on this patient, a woman aged 65, there were in the right lung five echinococci each the size of a pea; the liver was enlarged to four or five times its normal size. It contained an echinococcus as large as a child's head, with daughter-bladders the size of apples, two echinococci each as large as a fist, and one other the size of an apple. The omentum was adherent to the liver, and contained numerous echinococci varying in size from a hazel-nut to a walnut, and of these the smaller ones were degenerated. There were also echinococci in the appendices epiploicæ of the descending colon, in the pouch of Douglas, in the fundus of the uterus, and in the appendix vermiformis.

General Symptomatology.—The symptoms of echinococci are dependent on their site and size, and on the inflammatory and pressure effects on neighbouring viscera. In many situations, *e.g.* the liver, if the cyst does not interfere with any vital function, and does not injuriously affect the neighbouring viscera, it may produce no symptoms, and even when the size of a child's head may only be detected post-mortem. In other instances the hydatid, as it grows slowly, may have existed for two to thirty years, and be of considerable size before the patient seeks advice; yet the actual duration of the disease is always uncertain, for the date of infection can never be accurately determined. The condition is characterised by the slow and, as a rule, steady growth of the cyst forming a globular, painless, elastic fluctuating swelling, which is not accompanied by fever or cachexia. Fluctuation can only be determined when the echinococcus approaches the surface, and in such cases percussion may occasionally elicit the "hydatid thrill" or fremitus. This "thrill," however, can seldom be obtained, does not necessarily indicate the presence of daughter-cysts, and is not at all characteristic, as it may be elicited in ascitic fluid, or in any tense cyst such as a parovarian cyst. The echinococcus may attain an enormous size, and the pressure effects, which are similar to those of any other cystic tumour, are often very considerable. They vary, of course, as to the organs involved; echinococci of the liver produce embarrassment of the stomach, intestines, heart, or lungs; echinococci of the pleura or lung cause symptoms resembling those of pleuritic effusion or pulmonary tumour; echinococci of the pelvis cause symptoms referable to the rectum, bladder, etc. Erosion of bone may occur from pressure. If the disease affect the brain, spinal cord, or orbit, symptoms will arise even whilst the echinococcus is small.

The symptoms are also dependent on the degree of inflammatory reaction excited. If this be slight it results merely in the formation of a fibrous capsule, but it may, however, cause a chronic inflammation in the affected organ, *e.g.* hepatic cirrhosis, with consequent functional disturbance. Or the inflammation may proceed to suppuration with the formation of a localised abscess. This may rupture and so cause purulent inflammation of a serous cavity, or death may result from pyæmia.

In other cases, and usually in consequence of traumatism, a non-suppurating cyst may rupture. The gravity of this occurrence depends on the site into which the contents of the cyst escape. Rupture of a large cyst into the pleura, trachea, bronchi or peritoneum, is usually fatal; rupture into the alimentary or urinary tract, into the vagina or bile ducts, is much less serious, whilst rupture through the skin is the most favourable of all forms of rupture.

Echinococcus of the liver forms from 50 per cent (Neisser) to 66·9 per cent (Mosler and Piper) of all hydatids. It is usually single, situated in the right lobe, and of slow growth. If small it may cause no symptoms, seldom produces jaundice, though more often some cirrhosis, and the patient may be cured by death of the echinococcus as so often happens, or by rupture into the bile ducts. There may be dropsy, however, from pressure on the vena cava by a small hydatid, so situated as not to be detectable on examination. The cyst is, however, often of large size, may occupy the greater part of the abdomen, and is often adherent to other abdominal viscera which become displaced. Such a cyst produces discomfort, a dragging sensation in the abdomen, jaundice and dropsy being unusual. If the hydatid be of only moderate size there will be a visible swelling mainly in the right hypochondrium, smooth on the surface, usually yielding fluctuation, and sometimes a hydatid thrill. Over this swelling there is a dull note continuous with that of the liver. If suppuration occur in such a cyst the symptoms will closely simulate those of abscess of the liver; but if the cyst merely increase in size the abdomen becomes enormously distended, and pressure symptoms become well marked—dyspepsia, constipation, dyspnœa, palpitation, etc. Rupture of such a hydatid may occur into the intestines, with either a subsequent cure, or less frequently with suppuration in the cyst. Rupture into the stomach, intestines, or bile ducts is indicated possibly by some diminution in the size of the swelling, but more certainly by the occurrence in the vomit or fæces of portions of cyst wall, hooks, daughter-cysts ("skins"), etc., whilst rupture into the bile ducts may cause biliary colic. Rupture into the peritoneum is usually followed by fatal peritonitis, rupture into the vena cava causes death from pulmonary embolisms, whilst rupture into the kidney or through the skin is less serious.

If the echinococcus be situated towards the convex surface of the liver its enlargement occurs mainly upwards into the thorax. This variety is often spoken of as a "*subphrenic hydatid*," and in association with it there is often pleuritic effusion on the right side. The growing cyst displaces the diaphragm, right lung and heart, or may grow through the diaphragm, and more or less fill the right pleura.[1] The symptoms and signs of echinococci extending into the thorax from the liver are chiefly those of pulmonary hydatid disease.

Multilocular Echinococcus.—If symptoms be caused, as is not always the case, they differ from those of ordinary echinococci of the liver. In the former condition, which has never yet been seen before puberty, the first symptoms are mainly gastric, and are followed by jaundice which is usually very severe. The liver becomes much enlarged, is firm, hard, and often irregular on the surface, whilst it is not much altered in shape, and is not tender. There is often progressive enlargement of the spleen. The state of nutrition and general health of the individual remain remarkably good for a long time. At a later stage there are often hæmorrhages from various sites, and some fever from degeneration at the centre of the diseased area, and towards the end there is often very marked ascites. Multilocular echinococcus has been mistaken for carcinoma or hypertrophic cirrhosis of the liver, but it mainly occurs between the ages of twenty-seven and fifty, develops slowly, and does not cause cachexia.

Pulmonary Echinococci.—1. In the lungs (7·4, Neisser). The echinococcus is usually single, may be situated in any part, but more often on the right side than the left (25 : 11, Neisser), and is as a rule in the lower lobe. The adventitious capsule, as is always the case when the echinococcus is well protected, is extremely thin, and the cyst not infrequently occupies the greater part of one-half of the thoracic cavity. The pressure effects on the heart and lungs are slowly developed, for the cyst grows slowly and insidiously, and the patient who is apparently in good health merely suffers from dyspnœa on exertion. The physical signs are those of a solid tumour or a pleuritic effusion—impairment of movement, possibly bulging of the chest wall, dulness, with absence of vocal fremitus, breath sounds and resonance. The cyst while still of moderate size often ruptures into the bronchi.

Reid narrates the case of a man, æt. 37, who had never previously been ill, and who whilst in perfect health was suddenly seized with a violent fit of coughing and expectoration of over half a pint muco-pus. There was a patch of dulness below the angle of the left scapula with absence of breath sounds. The sputum contained abundant hooklets. The patient was operated on and recovered.

In some cases there is fever from secondary pleurisy, and if there be hæmoptysis too from rupture of the cyst, the symptoms are not unlike those of phthisis.

In spite of rupture into the bronchi there may be neither hooks nor portions of cyst wall detectable in the sputum.

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[1] Lenhartz has reported a case where by means of the Röntgen rays the diaphragm was found to bulge upwards to the level of the second rib (*Münch. med. Wchnschr.* 1899, p. 1696).

Reid records the case of a man, æt. 32, who six years previously had typhoid fever and pneumonia. The patient had ever since suffered from cough; he had repeated violent attacks of coughing with expectoration of large quantities of frothy, watery, slightly blood-stained and somewhat offensive sputum. Microscopic examination gave no evidence of hydatid disease; the pulmonary hydatid finally began to suppurate, and the patient recovered after operation.

In cases where a hydatid of the liver has perforated into the lung, the sputum may be of an ochre-yellow colour. Lenhartz in such a sputum found cholesterin, bilirubin and fatty granules.

2. In the pleura (1·9 per cent, Neisser). The echinococcus is either primary or else secondary owing to it invading the pleura from elsewhere, usually from the liver. The pleura itself usually constitutes the adventitious capsule. The signs are those of a pleuritic effusion, but the dulness is often localised, with its upper limit dome-shaped, and is not altered by change of position; whilst suppuration of the cyst produces symptoms similar to those of empyema.

A patient suffering from a pulmonary echinococcus may be cured by death of the parasite, rarely by rupture into the bronchi or alimentary tract, or rupture through the parietes. The disease is often fatal, death resulting from rupture into the bronchi or pulmonary artery, tuberculosis or gangrene of the lungs, or marasmus.

Echinococci of the Urinary System.—The kidney is affected in 8·9 per cent (Neisser)—2·8 per cent (Mosler and Peiper) of all cases of hydatids. The echinococcus is usually unilateral and situated primarily in the cortex, but as the cyst increases in size the whole kidney may become involved, and a large, firm, smooth swelling may be produced in one or other side of the abdomen. Symptoms may be entirely absent, and the condition may last for fifteen to twenty years, and not cause much inconvenience. The cyst may, however, attain an enormous size when there will be pressure symptoms, and adhesions will as a rule form between the cyst and the bowel, diaphragm, bladder, or spleen. Rupture of the cyst into the pelvis of the kidney may occur at any time, and is a somewhat favourable indication. Manasse has collected 51 cases from the literature where such rupture occurred. A case recorded by Wiesinger furnishes a good illustration. The patient had for two years a hydatid of the left kidney. He fell on his left side, the cyst ruptured, and blood and numerous hooks appeared in the urine. In consequence of rupture the abdominal swelling may diminish somewhat in size, and there is often at the same time renal colic. The urine rarely contains blood, but becomes cloudy or milky, contains pus cells, various crystals, hooks, portions of membrane, small daughter-cysts or brood-capsules, the latter being seen as transparent globules no larger than grape stones. The escape of cyst contents per urethram may continue for only a few days, or may recur from time to time for more than twenty years. By cystoscopy it was possible in Manasse's case to ascertain that the orifice of the right ureter was enlarged to four times its normal size, and that thence the hydatids entered the bladder. If symptoms of pyelitis or suppuration of the cyst should occur the condition becomes more serious.

Echinococci of the Bladder.—Manasse cites two cases, one of them having symptoms of severe cystitis and passage of hydatids per urethram. The latter symptom is, he says, the only characteristic sign of echinococci of the lower urinary passages in general, although there may be difficulty in micturition and defæcation, and although one may detect a fluctuating swelling. Manasse states that it is often difficult to determine whether the cyst is a primary hydatid of the bladder, or a hydatid which has developed in the subperitoneal connective tissue above the prostate, and which has become adherent to and possibly ruptured into the bladder.

Echinococci of the Spleen.—Three per cent (Neisser), 2·87 per cent (Mosler and Peiper). Trinkler in 1894 collected from the literature seventy cases occurring during the previous fifteen years. The cyst usually attains a large size in the course of time, and there is not infrequently an echinococcus in some other organ. The spleen itself is usually atrophied, less frequently hypertrophied. There are no special symptoms produced, but there is eventually a large fluctuating swelling which is either confined to the left hypochondrium or extends into other regions of the abdomen too. Or the hydatid grows upwards into the thorax, causes dyspnœa and other pulmonary symptoms, and may eventually rupture into the left pleural cavity or bronchi. Examination of the blood often shows merely a slight degree of anæmia, and the leucocytes are normal both as regards their total number and their relative proportions.

Other Abdominal Echinococci.—(i.) *Peritoneum.*—The hydatids are usually multiple, possibly because of previous rupture or tapping of a cyst in the liver. (ii.) *Mesentery or omentum* is rarely affected alone. A single large cyst usually forms a swelling towards the middle line of the abdomen. There may be no symptoms till the cyst suddenly ruptures and causes peritonitis, or until the cyst suppurates, when there may be symptoms of acute intestinal obstruction as in a case of Stirling's, where the patient awoke one morning in great pain, and then for the first time noticed a swelling in the epigastrium. (iii.) *Pancreas.*—No definite case has been hitherto recorded. Subbotic reports the case of a patient, æt. 17, where the clinical features were those of a pancreatic cyst, but at the post-mortem it could not be ascertained whether the hydatid had developed from the pancreas or the mesocolon. (iv.) *Lumen of the Intestine.*—There is as yet only one case in the literature. In this case, one of multiple echinococci, Subbotic states that in the interior of the appendix there were three echinococci varying from a cherry-stone to a cherry in size, and also a degenerated echinococcus as large as a bean. The cysts had apparently caused no symptoms. (v.) *Primary Retroperitoneal Echinococci.*—The symptoms are very indefinite, and an accurate diagnosis before operation is almost impossible.

Karewski records two cases: (*a*) A woman, æt. 35, suffered for years from symptoms of lumbago without definite ascertainable cause until a swelling eventually appeared in the region of the left kidney. Operation revealed a suppurating hydatid which had developed behind the descending colon below the kidney. (*b*) A man, æt. 55, had suffered for many years from sciatica, and a large swelling formed with accompanying emaciation and fever. The condition was diagnosed as a retroperitoneal abscess, and was in fact a suppurating hydatid cyst.

Pelvic Echinococci are not common. In the female sex they are usually situated in the broad ligament, ovary, uterus, or pouch of Douglas, and an exact diagnosis can hardly ever be made unless the cyst rupture externally. A case affecting the Fallopian tubes is recorded in vol. iii. p. 442.

Echinococci in Bones.—Targett in 1894 stated that there were records of 76 cases. The echinococcus develops either in the medullary canal or in spongy bone. It is as a rule, but not invariably, the exogenous variety which is found. Brood-capsules and hooks are rarely seen, the echinococcus is not surrounded by a true fibrous capsule, and slowly infiltrates the bone. The medullary canal becomes distended, egg-shell crackling can sometimes be elicited, and the bone is very liable to be fractured. The condition is usually painless, the surrounding soft tissues are often invaded, so that at some part or other a cystic swelling approaches the surface, and the disease in the later stages is often complicated by periostitis and necrosis.

Stirling (1896) relates that a man, æt. 43, had for five years noticed a slight swelling of the left thigh near the great trochanter. Since 1880 this thigh had been fractured by very slight violence five times. The patient was a healthy-looking man, well nourished, and had not suffered much pain, but the swelling had increased rapidly. The left thigh was found to be greatly enlarged in its whole extent, but especially at the upper part. The swelling was uneven and elastic in some places, especially at the inner and upper part of the thigh, where there seemed to be a distinctly cystic condition. Over the great trochanter there was a large, apparently solid tumour. There was no pulsatile movement and no lymphatic enlargement. The hydatid suppurated, the patient died, and at the post-mortem there was a large mother-cyst lining the cavity in the bone.

The symptoms of hydatid disease in the vertebræ may simulate those of Pott's disease; hydatid disease of the cranial bones produces a bulging of the outer table, and may at the same time cause signs of cerebral tumour.

Echinococci of the Nervous System.—Sixty-eight cases of echinococci in the *brain* are recorded by Neisser. The cyst may develop in the meninges, cortex, or central white matter. It is usually single and of limited size, being as a rule as large as a nut or an orange. The adventitious capsule is either poorly developed or entirely awanting. The cyst grows slowly, and produces the general signs of a cerebral tumour, with localising symptoms according to its site. Sudden death may occur at any time from rupture of the cyst, but probably both the prognosis and the results of treatment are more favourable than in many other forms of cerebral tumour. Echinococci in the cerebellum are very rare, in the medulla and spinal nerves they are almost unknown. In the spinal canal about 14 cases have been recorded, and the cyst usually invades the dura mater from without. Blaschek finds that there are 59 cases of echinococci in the orbit on record; the cyst causes gradual exophthalmos and amblyopia, and a fluctuating swelling may sometimes be found between the orbital margin and bulb.

The heart is rarely affected. According to Roche there were in 1897 only 45 cases recorded, and in the case which he himself records there was sudden death as so often occurs. Hydatid disease in arteries, veins, parotid, thyroid, muscles, subcutaneous tissues, and other sites is exceedingly rare. Gerulanos narrates a case where the muscles were affected with no less than 100 echinococcus cysts, varying in size from a lentil to that of two fists. A single echinococcus in the muscles is more common and may be mistaken for a lipoma, malignant tumour, or abscess.

Multiple Echinococci.—The symptoms vary according to the number, site, and size of the cysts. The liver is often enlarged, and there may be great abdominal distension, with fluctuating swellings in various parts of the abdomen, or more rarely in other parts, *e.g.* the axilla. Moore records such a case:—

The patient, æt. 38, had suffered occasionally from severe abdominal pains for a twelvemonth, and for the last seven months the abdomen had steadily increased in size. The patient had never any vomiting or jaundice. A number of rounded movable swellings could be felt all over the abdomen, but there was no ascites or hepatic enlargement. He was operated on four times, 47 cysts being dealt with in all, whilst at the first operation the whole omentum was seen to be studded with cysts varying in size from an orange to a hazel-nut. The patient made a good recovery.

DIAGNOSIS.—The apparently good health and unimpaired nutrition of the patient, the history of the slow yet steady growth of the tumour, and the presence of a cystic swelling, are important. The diagnosis, however, must be mainly made by exclusion. The chief conditions from which hydatid disease of the liver has to be distinguished are distended gall-bladder, hepatic abscess, various hepatic tumours, subphrenic abscess, and pleuritic effusion. The initial site of the swelling should be ascertained. If this prove to have been, say, the right or left hypochondrium, pelvic hydatids can be excluded. If the swelling commenced towards the middle line, and is very movable, but does not move on respiration, the cyst has possibly developed in the omentum. If the swelling started in the left hypochondrium it may be impossible, until the abdomen is opened, to say whether the cyst belongs to the left lobe of the liver or the spleen. Echinococcus of the kidney develops behind the colon. Hydatid disease in the pelvis can in most instances only be suspected; the diagnosis must be made at the time of operation; similarly with cerebral and spinal echinococci. Multiple echinococci in the abdomen may be mistaken for tubercular peritonitis. The special points as to pulmonary echinococci are the copious sputum with its peculiar characteristic microscopic appearances, the localised area of dulness, and in most cases the absence, after repeated examination, of tubercle bacilli. A cyst in the lower lobe of the left lung may be indistinguishable from one of the spleen. To the Röntgen rays, all echinococci, not only those with calcified walls, are comparatively non-transparent, and as regards the size and extent of some abdominal echinococci, skiagraphy has been claimed to yield more information than ordinary physical examination. It may be useful, too, in locating pulmonary echinococci (Levy-Dorn and Zadek, *Berl. klin. Wochns.*, 1899).

If the cyst have ruptured, the diagnosis is usually comparatively easy. Otherwise a definite diagnosis is often impossible without an exploratory puncture. As fluid in some varieties of hydronephrosis and cerebro-spinal fluid closely resemble hydatid fluid chemically, the absence of albumin is not so characteristic of the latter as scolices, hooks, etc., the presence of which enables a positive diagnosis to be made. Tapping, however, unless it can be followed by immediate operation, should never be performed. For it is sometimes followed by serious symptoms, such as shock, syncope, dyspnœa, vomiting, convulsions or high fever. Tapping is not infrequently followed by suppuration in the cyst, and the puncture of an abdominal hydatid may bring about the development of multiple echinococci of the peritoneum, or the trocar may wound some important structure such as the portal vein. An urticarial eruption, termed the "hydatid rash," may also appear after puncture, coming on rapidly, and involving the abdomen, thorax, face, and arms. The rash persists for 24 to 72 hours, and is usually accompanied by severe pruritus. These symptoms are thought to be due to the absorption of some special toxic constituent of hydatid fluid. The toxic substance is capable of producing a great fall of the blood-pressure in the systemic arteries (Roy), and is regarded as a ptomaine (Mourson and Schlagendhauffen), or as a toxalbumin (Viron). The hydatid rash has been experimentally produced in the human subject by injection of hydatid fluid (Debove), but Graham found almost no disturbance produced by the injection of the fluid into dogs.

In the bones hydatid disease is most readily mistaken for sarcoma; but in the former there is a history of long-standing disease, the swelling is not pulsatile, and the lymphatic glands are not affected.

Prognosis.—Hydatid disease is always a serious condition because of the risk of suppuration or rupture of the cyst, the former being the danger of most frequent occurrence. The prognosis varies, however, according to the site and size of the cyst, on its degree of adhesion to neighbouring viscera, and on the ease with which it can be reached by the surgeon. Signs of degeneration and death of the parasite are favourable.

PROPHYLAXIS.—In a district where the disease is endemic persons should be careful how they handle dogs, and if possible the number of dogs should be reduced to the necessary minimum. Dogs should not be allowed access to the water-supply, all drinking-water should be boiled, and uncooked vegetables thoroughly washed before being eaten. No dogs should ever be admitted to the slaughter-houses, and all offal containing hydatids should be destroyed, so that dogs may have no opportunity of eating it. The administration of anthelmintics to dogs has been recommended, as their use has been found of importance in reducing the frequency of "staggers" in sheep, whilst in Victoria one is advised to bury or throw boiling water over the exposed fæces of dogs, and to frequently purify with boiling water the kennels and the ground around them.

TREATMENT.—Although hydatids grow very slowly, and although many patients may undergo cure by spontaneous death of the parasite, yet every patient suffering from the disease should be treated, as his life may be at any time endangered by suppuration or rupture of the cyst. Surgical interference is the only mode of treatment in general use at the present time, and the methods of operation are as varied as are the characters of different hydatids. The procedure to be employed must therefore be that which appears best adapted to the individual case.

I. Incision of the cyst with drainage of the contents (marsupialisation of the French writers). A hydatid of the liver may be taken as an example. It may be dealt with (*a*) *by operation in one stage* (Lindemann). The abdominal wall is incised, the cyst exposed and brought up to the wound; swabs are applied to prevent any fluid passing into the peritoneum when the cyst is opened; the adventitious capsule is sutured to the edge of the parietal wound; the cyst is opened; the contents are thoroughly evacuated by irrigation, aided by gentle traction with forceps on the true cyst wall or daughter-cysts, and a large drainage-tube is inserted. In a favourable case healing will be complete in six to ten weeks. In Landau's modification the cyst is exposed, and is then tapped before the adventitious capsule is sutured to the parietes. (*b*) *Operation in two stages* (Volkmann). At the first operation the cyst is exposed and the wound packed with gauze. Adhesions will form between the serous surfaces, and about a week later the cyst is opened, emptied, and drained as in Lindemann's operation.

Indications.—Suppuration of the cyst, or any large cyst which from its size or configuration, or because it contains numerous daughter-bladders, cannot be completely and thoroughly emptied.

Disadvantages.—It is not uncommon for healing to be slow; a chronic fistula discharging bile may remain, the patient is exposed to the risk of septic infection, and finally a bad scar may result, with the possibility of subsequent ventral hernia.

If there are no peritoneal adhesions, which is rarely the case in suppurating hydatids, operation in two stages is claimed by some to be safer than Lindemann's operation, but in the former method the patient has to undergo a second operation, and the formation of adhesions cannot always be relied on even a fortnight after the first operation. There is in fact no advantage gained by operating in two stages, provided that in Lindemann's operation requisite precautions are taken to prevent the entrance of hydatid fluid into the peritoneum.

II. Evacuation of the cyst and closure of the abdominal wound.

(*a*) *Modified Lindemann's Operation.*—After the cyst has been exposed and tapped, the adventitious capsule is firmly sutured to the edge of the parietal wound, the cyst is incised, the mother-cyst, daughter-cysts, and other contents are cleared out, the wall of the sac being thoroughly cleansed and dried with swabs. The edges of the incised adventitious capsule are then sutured together, and the parietal wound is closed. (*b*) *Intraperitoneal Treatment.*—The cyst is exposed, incised, thoroughly cleared out so as to remove all the mother-cyst, daughter-cysts, etc., and the sac is dried out with swabs. The edges of the incised adventitious capsule are sutured, the sac is dropped back into the abdomen, and the parietal wound is closed. There are various modifications of this operation. Delbet recommends the passing of sutures through the thickness of the walls of the sac so as to bring the latter in apposition. Posadas (*Rev. d. Chir.* 1899, xix.), however, not only regards Delbet's method as useless because the walls come naturally in contact without sutures, but as dangerous and impracticable. Tuffier scrapes the cyst wall and washes out the cavity with sublimate; others fill the cavity with iodoform glycerine.

In all these methods the wound is closed and no drainage employed. This is the procedure recommended of late by many writers besides those already mentioned. The procedure is applicable to non-suppurating cysts, to those where the whole of the mother-cyst and all daughter-cysts can be completely extirpated, and also, as stated by Posadas, to cysts which, by their adhesions to important viscera, or by their inclusion in such organs as are physiologically indispensable, cannot be completely removed by methods described below.

The healing of the wound by first intention obviates the intractable fistulæ, protracted convalescence, and other disadvantages of Lindemann's operation. One of the dangers of these methods, however, is that there may be recurrence of the disease owing to incomplete evacuation of the cyst; daughter-bladders, for example, may be left behind. Büdinger relates such a case. The liver was the seat of an echinococcus the size of a man's head, yet the patient had been operated on six years previously and regarded as cured. The special danger of the intraperitoneal method is that suppuration may occur in the sac and cause peritonitis, whereas should the sac suppurate, or its cavity fill up with bile after the "modified Lindemann's operation," the removal of a suture and a drain are all that is required (Stirling). Posadas records thirty-six cases treated by the intraperitoneal method. In twenty-eight cases there was healing by first intention; in three cases suppuration occurred, drainage was required, but the subsequent cure was rapid, and in five instances there was a fatal termination.

III. Enucleation may be performed in certain cases, *e.g.* a cyst in the liver or subcutaneous tissues, provided it be not larger than an ostrich's egg. The adventitious capsule is cautiously incised, and the echinococcus within is removed without a drop of fluid escaping (Posadas). Removal of the whole cyst *en masse* may be possible if the cyst be free, without adhesions, and with a pedicle, *e.g.* certain cysts of the omentum (Deguy); or extirpation of the cyst together with the organ—spleen, kidney, etc.—in which it has developed may sometimes be carried out.

Special points in the treatment of (*a*) *Subphrenic Hydatids.*—Access to the cyst has often to be obtained through the thoracic wall with resection of portions of one or more ribs. The pleural cavity will in many cases be obliterated at the level of the incision, but if not, the pleural surfaces are to be stitched together and the cyst fixed to the edge of the wound before the latter is opened. If irrigation and drainage are indicated a counter-opening below the costal margin is useful. (*b*) *Pulmonary Echinococci.*—Tapping is exceedingly dangerous because of the risk of drowning the patient with hydatid fluid. In one of Graham's cases the introduction of the needle led to the rupture of the cyst with consequent flooding of the healthy lung and sudden death. Operation is to be carried out on the lines indicated for subphrenic hydatids, except that as a rule no counter-opening is required. (*c*) *Echinococci of the Kidney.*—Most writers recommend incision with or without drainage. If the kidney appear to be entirely destroyed the advisability of nephrectomy may be considered. Sserapin states that of thirteen patients in whom nephrectomy was performed five died. (*d*) *Echinococci of the Spleen.*—The mortality of treatment by incision and drainage is stated by Roche to be 30 per cent. Splenectomy is facilitated by atrophy of the spleen and mobility of the tumour (Hahn), and Hartmann knew of eleven cases thus treated with only two deaths. (*e*) *Echinococci in Bones.*—Incision and scraping of the cyst with incision and drainage of abscesses may be tried, but suppuration is a serious occurrence, and if the disease be extensive amputation of the part is advisable.

Other methods of treatment must be mentioned, though they are now seldom if ever employed. I. Evacuation by aspiration and injection of (*a*) 25-30 c.c. of a 1-1000 solution of corrosive sublimate, which is left in (Hanot); (*b*) 100-500 c.c. of the same fluid, and evacuation of the same after leaving it in for ten minutes (Debove, Mesnard). Partial evacuation of the hydatid fluid (20-50 c.c.) and injection of a similar quantity of sublimate solution (Bacelli), or of a 1-5 per cent solution of silver nitrate (Berrucio). Iodine, carbolic acid, alcohol, etc. have also been used instead of sublimate. All of these methods are associated with the dangers of simple tapping.

II. Tapping was formerly the recognised method of treatment, but the associated dangers are so great that it should never be performed under any circumstances unless it immediately precedes operation by incision. In some instances the withdrawal of fluid does cause the death of the parasite, in other instances the fluid slowly reaccumulates, whilst finally in other cases the signs of refilling are simulated by the comparatively rapid growth of an echinococcus closely adjacent to the one which was tapped.

III. Electrical treatment bears with it the dangers consequent on tapping, yields unreliable results, and has never been generally adopted. Boinet, recording in 1897 a case treated by electro-puncture, is the only writer who has of late advocated the employment of electricity.

IV. Obsolete methods of historic interest. Such are the production of adhesions between the cyst and the parietes by the use of caustic pastes, or by the insertion into the cyst of two trocars and cannulæ (Simon's method), with subsequent opening of the cyst; and, lastly, medicinal treatment, which has never proved to be of any real value.

LITERATURE.—The reader should consult the Australian literature for details of illustrative cases and treatment. The names of most of the writers referred to in the preceding article are given in the following list:—**General**: Cobbold. *Parasites, a Treatise on the Entozoa of Man and Animals*, 1879.—Neisser. *Die Echinoc.-Krankh.* 1877.—Mosler u. Peiper. *Thiersche Parasiten*, Nothnagel's *Spec. Path. u. Therap.* vi. 1894.—Verco and Stirling. Allbutt's *Sys. of Med.* ii. 1897. **Development**: Leuckart. *The Parasites of Man.* Trans. by Hoyle, 1886. **Hydatid Fluid**: Roy. *Lancet*, 1897, i. p. 121.—Viron. *Arch. d. méd. expérim.* 1892, p. 136. **Geograph. Distribution and E. Multilocularis**: Posselt. *Die geograph. Verbreit. d. Blasenwurmleidens*, 1900. **Liver**: Murchison. *Hydatid Tumours of the Liver*, 1865; *Clin. Lect. on Dis. of the Liver*, 1877. **Pulmonary**: Reid. *Intercol. M. J. Australas.* 1897, p. 608. **Urinary System**: Manasse. *Centralb. f. d. Krankh. d. Harn u. Sex. Org.* 1898, pp. 597, 654.—Houzel. *Rev. de Chir.* 1898, pp. 689, 811. **Spleen**:

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Hydroa. *See* DERMATITIS HERPETIFORMIS.

Hydrocele. *See* SCROTUM, TESTICLE.

Hydrocephalus.

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Definition.—Hydrocephalus signifies a collection of fluid in the cavity of the cranium. The fluid may be situated between the dura mater and the brain, *i.e.* external or subdural hydrocephalus; or it may occupy and distend the ventricles, *i.e.* internal hydrocephalus.

Hydrocephalus may be acute or chronic, and the acute or chronic variety may be external or internal. The common form is chronic and internal, and is the only form which needs a detailed description here.

ACUTE HYDROCEPHALUS.—This variety, whether external or internal, is the result of inflammation of the pia mater; it is doubtful if an acute hydrocephalus occurs under any other circumstances. Tuberculous meningitis is the chief cause; it is common to find at post-mortem examinations of those dying of this disease a large excess of fluid in the subdural space, and perhaps several ounces occupying the ventricles.

CHRONIC HYDROCEPHALUS.—Chronic external hydrocephalus is a comparatively rare condition, and is usually secondary to some brain lesion. Whenever the brain, or a portion of the brain, undergoes shrinkage, a collection of fluid takes place occupying the subdural space corresponding to the atrophied part. A considerable amount of fluid may be present in some cases, as for instance when one-half of the brain has undergone partial atrophy. A collection of fluid may also be present in the subdural space in cases of chronic or pachymeningitis. Subdural fluid may be found in cases of internal hydrocephalus, in which the fluid has burst through some part of the brain and drained into the subdural space.

CHRONIC INTERNAL HYDROCEPHALUS.—In the common and important form of hydrocephalus the fluid is poured out into and gradually distends the lateral ventricles; as the fluid increases the head enlarges, the bones of the skull open out, the fontanelles bulge; there may be mental dulness and perhaps rigidity of the limbs.

Etiology.—Under normal conditions the amount of cerebro-spinal fluid in the ventricles and subarachnoid space is small. According to Leonard Hill "the living brain with its circulating fluid almost entirely fills the cranium, and the fluid that moistens its surfaces is little more in amount than the synovial fluid in a joint." It is true that, post-mortem, several ounces of fluid will drain away from the cranial cavity and spinal canal after the skull has been removed and the dura mater incised, but it is probable that a certain amount of this is poured out during the act of dying, when the cerebral veins and sinuses are gorged and distended. During life under normal conditions any excess of cerebro-spinal fluid secreted in the lateral ventricles drains away *via* the third ventricle through the aqueduct of Sylvius into the fourth ventricle, and thence into the subarachnoid space through the several openings in the arachnoid membrane reflected from the cerebellum over the fourth ventricle (foramina of Majendie and Luschka). The cerebro-spinal fluid thus making its escape from the lateral ventricles into the subarachnoid space is absorbed by the veins. It is easy to understand if the "way out" of the lateral ventricles be occluded, fluid will be ponded up in the ventricles, and if the resistance to expansion be small, the amount of fluid will gradually increase and dilate the ventricles. A posterior basal meningitis is very liable to produce a matting of the structures in the neighbourhood of the fourth ventricle, and consequently occlusion of the foramina of Majendie and Luschka. The same result will follow if the aqueduct of Sylvius be blocked by a tumour, as pointed out by Hilton. Another mechanical form of chronic hydrocephalus is secondary to some obstruction of the veins of Galen by a tumour of the cerebellum or neighbourhood. The tentorium becomes stretched and the straight sinus compressed. As the veins of Galen drain the choroid plexuses and intraventricular veins, any obstruction to the former will lead to an excessive pouring out of fluid in the ventricles. It must be admitted, however, that the greater number of cases of chronic hydrocephalus, especially those which are congenital or which are first noticed during the first few months of life, are not the result of meningitis or tumour. In some of these cases a chronic inflammation (? syphilitic) of the choroid plexuses has apparently taken place, but in others no satisfactory explanation is forthcoming.

From the above considerations we may divide the cases of chronic hydrocephalus into two groups; the larger group includes the cases which in the present state of our knowledge we call *primary*, and which are either congenital or occur during the first months of life, and the secondary cases which follow a cerebral tumour or meningitis, and which may arise at any time of life and occur both in children and in adults. The symptoms in both groups, making allowance for age and the other lesions present, will be very much the same.

Symptoms.—The most marked and distinctive symptom of chronic hydrocephalus is progressive enlargement of the head. At birth the circumference of the normal head is roughly 14 in., at six months 16½ in., at a year old 18 in., at two years 20½ in. In well-marked cases of hydrocephalus the circumference may be increased by 2 or 3 in., and in extreme cases to as much as 8 or 10 in. In one of the author's cases the head measured 28 in. in an infant a year old. The shape of the head is globular, the cranium enlarging in the transverse more in proportion than in the longitudinal direction; the enlarged round cranium contrasts markedly with the face, the forehead and occiput strikingly overhang the face and neck. The fontanelles are widely open and bulge outward; they have a tense, elastic feel on pressure with the finger; the sutures are separated perhaps half an inch in some places. The scalp and skin over the forehead are stretched and shiny, and the superficial veins distended. The infant is unable to hold up its head, or indeed to have much control over its movements; it is usually mentally dull and takes but little notice of its friends, but this is by no means the rule, as it is often surprising to note that a hydrocephalic infant with an excessively large head will smile and show signs of moderate intelligence. In the worst cases the infants' health suffers, it gradually wastes, and its emaciated body contrasts greatly with the enormous size of its head; life is only prolonged a few months.

In other cases the course is more chronic, and the infant's health suffers less; it continues to live month after month, and in some cases year after year. It is unable to sit up or support its head, its intelligence is poor, though probably able to understand a good deal of what is said to it; the limbs become weak, especially the legs, the deep reflexes are exaggerated, with a tendency to spastic rigidity. In the most extreme cases which live to be a few years old, the eyes are pushed forwards and downwards, there is divergent squint, and difficulty in closing the eyelids. The limbs become flexed and rigid, there is complete amentia, and possibly atrophy of the optic nerves.

In a few cases in which hydrocephalus is not extreme, the head ceases to enlarge, and the skull may gradually close up. The child learns in time to walk and talk, but in the majority of cases the intellect never fully develops. Such children may be seen when a few years of age attending the outpatients of a hospital, with large heads and uncertain gait; or they may be found in asylums as "hydrocephalic idiots"; or they drift into the workhouse as they are unable to earn their own living.

In a still smaller number of cases complete recovery appears to take place. The head ceases to enlarge, the fontanelles become less bulging, and in the course of many months or longer the head may regain its normal proportions. Improvement or recovery can hardly be expected in any but the slighter cases. Some of these cases are the subjects of congenital syphilis.

In secondary hydrocephalus the symptoms are likely to be mixed up with those of the primary disease. Thus in the posterior basal meningitis of infants and children there will be the symptoms of meningitis, retraction of the head, etc., the fontanelle—if still open—will be tense and bulging, the skin of the forehead and scalp tense, and the circumference of the head increases in size. In these cases, if they survive, the enlargement of the head is seldom great; the hydrocephalus may perhaps be due to the obliteration of the foramina of Majendie and Luschka, or possibly to an intraventricular meningitis. When hydrocephalus occurs as the result of some obstruction to the veins of Galen, it is mostly at an age when the fontanelles have been closed and the sutures more or less firmly fixed than they are during infancy. In such cases the head will necessarily enlarge more slowly, and never reach extreme proportions, and in some cases there will be no enlargement at all. Perhaps the commonest cause of secondary hydrocephalus in children is a tubercular tumour of the cerebellum. As the tumour-growth proceeds the tentorium is stretched, the straight sinus and vena Galeni are obstructed, and fluid gradually accumulates in the lateral ventricles. In the more severe cases in children below the age of puberty the head may slowly enlarge, and the parietal and frontal bones separate, but this takes place probably in the later stages when the growth is of large size and the child bedridden. In the early stages, and in the slighter cases, when there is no enlargement of the head, the diagnosis of hydrocephalus is well-nigh impossible; in any case to differentiate between symptoms produced by the hydrocephalus and those produced by a tumour of the cerebellum is very difficult. A collection of fluid in the ventricles will be likely, by compressing the internal capsule and neighbouring parts, to produce paresis of the legs with exaggerated knee-reflex. Some of the symptoms attributed to cerebellar tumour, such as uncertain and staggering gait, may be really due to the hydrocephalus which accompanies the tumour, and not immediately to the tumour itself. Hydrocephalus may be secondary to a sarcomatous tumour of the choroid plexus or blockage of the aqueduct of Sylvius by a tumour or clot of blood.

In certain cases in older children, in which the enlargement of the head is slight or not marked, chronic hydrocephalus has been found post-mortem, when death has occurred from some other cause. In such cases the course has been very chronic and suggestive of a slowly growing cerebral tumour, as there may be headache or more or less weakness of the limbs. On the other hand, vomiting and optic neuritis would be in favour of a tumour with or without hydrocephalus. Optic atrophy does, however, occur in some cases of chronic hydrocephalus apart from tumour. In these cases of chronic hydrocephalus the causation may be doubtful; possibly they may have suffered from congenital hydrocephalus and partially recovered, or they may be secondary to some slight basal meningitis.

In rare cases an escape of cerebro-spinal fluid from the nose, the fluid constantly draining away—much to the discomfort of the patient—has been practically the only symptom of chronic hydrocephalus.

Morbid Anatomy.—Normal cerebro-spinal fluid is clear and colourless as water, faintly alkaline, and of sp. gr. about 1005. It gives a slight opalescence with heat and acetic acid, and often a slight reduction of copper oxide when heated with Fehling's solution. The proteid present is globulin, not albumin, and the copper-reducing substance is pyrocatechin, not sugar (Halliburton). The fluid drawn off from many cases of hydrocephalus is identical with normal cerebro-spinal fluid; in other cases, as when it is the result of meningitis (posterior basal), the fluid is turpid and albuminous. The amount of albumin may be 0·3 per cent (Esbach) or more, and the specific gravity 1008. The amount of fluid present may vary from a few ounces to many pints. The changes in the brain consequent on a gradual distension of the ventricles will obviously be a compression and thinning of the brain substance which surrounds the ventricles. In extreme cases this is so great that the roof of the ventricles appears in places like a cyst with thin transparent walls. The fluid may burst through the corpus callosum or cortex. The floor of the ventricles is compressed and consequently flattened, and the internal capsule suffers compression. The aqueduct of Sylvius may be dilated and large enough to admit the little finger, while the floor of the 4th ventricle is also flattened by the pressure of the fluid. The choroid plexuses are usually normal in appearance, but a chronic inflammatory condition has been observed. The frontal, parietal, and occipital bones may be separated widely at the roof of the skull.

Diagnosis.—There can be no difficulty in making a diagnosis when the disease commences before the head has closed up. The gradual enlargement of the skull, the bulging fontanelles, and open sutures make recognition of the hydrocephalus easy. The most likely cases to present difficulty are those of rickety children, who, during their second or third year, have big heads with small bodies and limbs. The rickety skull is mostly flat on the top and square in outline, on account of the exaggerated frontal and parietal eminences; the superior fontanelle may be open, but not bulging, and the edges of the bones feel thickened. The sutures are closed; there are often shallow grooves corresponding to the transverse and longitudinal fissures. The brain in these cases is frequently larger than normal, but there is no hydrocephalus. Diagnosis is necessarily difficult when the disease commences after the skull has closed up; gradual enlargement of the head with opening up of the sutures are the important points. Chronic hydrocephalus by itself, or in association with a cerebellar or other tumour, and without enlargement of the head, cannot be diagnosed with any certainty. Headache, paresis of limbs, exaggerated knee reflexes would probably be present, but it is needless to say these symptoms by themselves would not be distinctive.

Prognosis.—This is always grave, whether the disease is primary or secondary. In the majority of cases which arise within the first few months the disease steadily progresses, and death occurs within the first two years; in a few cases life is prolonged a few years, but rarely beyond the sixth or seventh. In a minority of cases the enlargement of the head ceases, the skull closes up, but the head remains abnormally large. In a very small number of cases complete recovery appears to take place. This can only be expected in the early stages, and when the hydrocephalus is slight or moderate in degree.

Complications.—Congenital cases of hydrocephalus may be associated with other and somewhat similar lesions as spina bifida, meningocele, and encephalocele. In some instances there has been club-foot or hare-lip.

Reference has already been made to a leakage of cerebro-spinal fluid from the cavity of the skull through the cribriform plate of the ethmoid bone into the nasal cavity.

Treatment.—In all cases of primary hydrocephalus, especially in those cases arising during the first few months of life, no time should be lost in commencing antisyphilitic treatment. The prospect of cure cannot be said to be very brilliant, but a certain proportion of these cases are syphilitic, and cures have been apparently effected by mercury and iodides, and it is at least worth while trying if they are of service, especially in early cases. Half a grain to a grain of gray powder may be given twice daily with one to two grains of iodide of potassium. This treatment should be persevered in for many months. Apart from such treatment it cannot be said that drugs are of much use, and medicinal treatment becomes a treatment of symptoms.

THE SURGICAL TREATMENT OF HYDROCEPHALUS

Surgery has chiefly concerned itself with the treatment of chronic internal hydrocephalus or chronic distension of the lateral ventricles, and principally with those cases which are due to some cause other than cerebral tumour.

Occasionally need for surgical measures arises in cases which may be considered as examples of localised external hydrocephalus, where a collection of fluid is formed in some limited space either between the dura mater and the arachnoid or in the subarachnoid space so-called. Many of the instances of localised external hydrocephalus should rather be considered as examples of cysts resulting from blood extravasations or as passive collections of fluid poured out to replace an atrophied or shrunken brain. Though occasionally such conditions may require operative treatment, they hardly come within the scope of the present article.

Since the term acute hydrocephalus has been used as synonymous with acute meningitis due to tuberculosis or other conditions, the consideration of its treatment by operation must be briefly undertaken here, before the question of chronic internal hydrocephalus with which we are chiefly concerned is dealt with.

Acute Hydrocephalus.—If the evil effect of an acute meningitis, whether traumatic or simple or tubercular, was due entirely to pressure by a localised collection of thin fluid which could be readily drained away, its treatment by operation would be comparatively simple and probably satisfactory, the chief difficulty would arise in finding the position of the fluid, but it is obvious that other factors come into play, the fluid is only a part of the exudation, which may be mainly composed of lymph, and this by pressure upon important structures may render futile the attempt to give relief by drainage. Further, the inflammation may have involved the brain tissue and not merely the surface of the meninges, hence drainage in many cases cannot be expected to do more at best than give partial and temporary relief.

If, however, it is determined to try the effect of draining off the fluid, the attempt may be made either by trephining or puncturing the skull or by "lumbar puncture" (Quincke, *Centralblatt für Chirurg.* 1892); *i.e.* tapping the spinal theca in the interspace between the third and fourth lumbar vertebræ, and thus either directly or indirectly the fluid effusion is drawn off. The question of draining for a longer or shorter period must be decided by the nature of the fluid; if this is sero-purulent or purulent it would, no doubt, be wiser to make provision for drainage: if the object was merely to relieve a suddenly increasing intracranial pressure, the removal of a certain quantity of fluid at the time would possibly be sufficient. If the puncture was made for the purpose of clearing up a doubt as to the tuberculous nature of the disease by examination of the fluid, aspiration would be sufficient, and might also produce a beneficial effect analogous to that obtained by removal of the fluid from a tuberculous peritoneal cavity.

For the performance of "lumbar puncture" it is only necessary to render the skin and syringe aseptic, and then with or without local or general anæsthesia to pass the needle between the laminæ of the third and fourth lumbar spines slightly to one side of the middle line. The patient is made to stoop forwards, or the spine is fully flexed so as to widen the space as much as possible, and then the needle is passed gently in. If it enters to a sufficient depth and fluid is drawn off, it will be known that the theca has been tapped. If the needle impinges upon bone, its direction should be changed so that it passes below the edge of the lamina and enters the theca. After withdrawal of the needle the puncture may be sealed by a small collodium or celloidin dressing.

In a certain number of cases of meningitis, whether due to syphilis or tubercle or other cause, the ventricles become distended either with or without a subdural collection of fluid. Under these conditions the apertures leading from the ventricles to the sub-arachnoid space may be closed. A good many cases are on record where an operation has been done to drain away this intra-ventricular fluid as in chronic hydrocephalus. Thus Parkin (*Lancet*, 1st July 1893) trephined below the superior curved line of the occipital on the right side, opened the dura, and passed a bent probe beneath the cerebellum, thus setting free the fluid, which may be drained away either by a small tube, or better, by use of a bundle of horsehair. Parkin had a case in which a fatal result followed in sixteen hours. Ord and Waterhouse (*Lancet*, vol. i. 1894, p. 397) record a case of tuberculous meningitis, in which a very similar operation was successful, and the child, which was five years old, recovered after seventeen days' drainage.

A paper of Morton's (*Brit. Med. Jour.* 8, 4, 1893) on the anatomy and method of reaching the intraventricular fluid without traversing the brain substance may also be consulted.

Chronic Internal Hydrocephalus.—The surgical treatment of this condition may be limited to an attempt to check or even diminish the rate of secretion of the fluid by pressure applied to the head. This has been done by strapping the head circularly with plaister, or by the application of an elastic bandage. Though such a simple and obvious measure has been many times attempted, it does not appear that any good result has followed, and there is real danger of causing sloughing of the scalp by compression of the vessels between the bandage and the unyielding skull. Another method of treatment is by tapping the ventricle through one of the lateral angles of the anterior fontanelle. The middle line is to be avoided on account of the presence of the superior longitudinal sinus. The tapping may be done with a fine hollow needle, a small trocar, or an aspirator, and after a certain amount of fluid has been drawn off, the opening may be sealed, or drainage for a time may be provided for by leaving in a capillary tube. For this purpose Southey's trocars have been employed. With this method of puncture may be combined the use of pressure to try to prevent reaccumulation of the fluid. Hern (*Brit. Med. Jour.* 11th Nov. 1893) records six cases of repeated tapping, with improvement in five. A further step consists in the attempt to check intraventricular secretion by the injection of iodine or other irritating fluid.

Prof. Keen suggested making a trephine opening 1¼ inch above and behind the external auditory meatus, and then puncturing the brain with a needle directed towards a point 2½ inches above the opposite meatus. The ventricular cavity was reached at a depth of about 1¾ inch. In a case in which this operation was done, a small horse-hair drain was kept in the ventricle for fourteen days, and then a drainage-tube was substituted. Twenty-eight days later a similar operation was performed on the other side, and a tube passed into the ventricle. The child died on the forty-fifth day. Two other cases died four days and four hours respectively after operation. The first case was one of cerebellar tumour (*vide* Jacobson, *Operations of Surgery*, from whom the above is taken).

It is obvious that the measurements given above are only very vague guides, inasmuch as they appear to refer to adult heads or those in which the ventricles are not dilated. It is clear that in a dilated ventricle with a thinned brain the depth from the surface could be nothing like 1¾ inch. It should also be noted that in a case in which the hydrocephalus was a mere secondary effect of a cerebellar tumour no good result could be expected.

Mayo Robson (*Brit. Med. Jour.* 1890, vol. ii.) trephined and punctured the ventricle in a case supposed to be one of basal meningitis resulting from ear disease. After failing to find pus he withdrew from the ventricle ʒvj. of clear fluid. The puncture was made in the posterior extremity of the second frontal convolution. No drainage was used. The child recovered. This case, again, can hardly be taken as a guide to the treatment of ordinary cases of chronic hydrocephalus. Mr. Robson points out that mere leakage or drainage away of cerebrospinal fluid is not necessarily fatal, a fact well established by the records of cases of fractured skull, with laceration of brain involving the ventricle. He gives Prof. Fraser's directions for reaching the different cornua, though these are perhaps hardly necessary, since in any case likely to call for treatment a puncture through the cortex at any point of the upper part of the hemisphere would reach the dilated ventricle. Care will, of course, be taken to avoid the motor area. Robson records another case of ordinary hydrocephalus in which he operated, but the drainage-tube slipped out and the child died.

Bruce Clarke, in the discussion on Robson's paper, mentions a case of chronic meningitis with subdural collection of fluid (external hydrocephalus) which he drained. The patient died in four days. Kendal Franks tapped a ventricle with temporary improvement, but death occurred in four days, which seems about the usual time. In this case the ventricle, after death, contained but little fluid, but there was a purulent basal tubercular meningitis. Wheelhouse had a similar experience.

Jacobson, to whom we owe several of these references, quotes a case of Broca's, in which drainage appears to have been successful up to the fiftieth day, when the wound was healed and the child discharged.

The operation of Parkin already described was performed on a child of eleven months with chronic hydrocephalus. The child was becoming comatose, but after eighteen days' drainage recovered and became more intelligent than it had been before the onset of the acute symptoms. The plan would apparently be applicable to cases in which the foramen of Majendie was closed by adhesions.

Sutherland and Watson Cheyne (*Clin. Soc. Transactions*, vol. xxxi.) devised and carried out a plan of draining the ventricles into the subdural space. An opening was made in the left lateral angle of the anterior fontanelle (the child was six months old), and a bundle of fine catgut tied together at one end was passed into the subdural space, the other end of the bundle was by means of sinus forceps carried into the lateral ventricle, and the wound closed. The size of the head diminished steadily, and for a time the child did well, but a convulsion came on after the first month, and after nine weeks it began to go down hill, and died three months after operation, with rapid wasting, screaming, and opisthotonos. The shrinkage in size of the head was remarkable, and good drainage had been apparently established. The principle of the operation in establishing drainage and preventing intraventricular pressure is good, and the operation is apparently very simple. The length of drain should be only sufficient to project $\frac{1}{4}$ inch into the ventricle. The child was syphilitic, but the plan, though not ultimately successful, is more promising than the other methods devised, except, perhaps, that of Parkin. In this case, as in so many other operations, craniectomy, simple tapping, and so on, the temperature rose high at first, nearly to 106°, but subsided gradually, and was clearly not due to septicity.

Starr (*Brain Surgery*) records some other cases of operation for hydrocephalus. In 1898, Bruce and Stiles (*Brit. Med. Jour.*, 29th Jan. 1898) treated a congenital syphilitic child of thirteen years suffering from basal meningitis and severe and increasing symptoms of intracranial pressure by an operation like that of Parkin's, except that the opening was made in the middle line, and included the margin of the foramen magnum. On separating the amygdala a large quantity of fluid escaped and continued to drain away. The child improved, but died after fifteen days of hyperpyrexia, possibly the result of renal disease.

On the whole it is probably best, in any case with progressive symptoms, to try either Cheyne's or Parkin's method.

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Hydronephrosis. *See* Kidney.

Hydropathy.

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See also Balneology, Mineral Waters.

Hydropathy, or treatment by water, is a most patent method either for good or ill. It covers an enormous amount of ground; the uses of water—hot, cold, and frozen—internally and externally, by the aid of various mechanical methods, and in both acute and chronic disease. The drinking of water is absolutely necessary; without it life would be impossible. The human body contains about 59 per cent of water; there is a constant loss going on through the agency of respiration and the various secretions which must be made good. The internal functions of tissue change and the expulsion of waste products depend largely upon water.

The drinking of cold water lowers for the time the temperature as taken in the axilla or rectum, reduces the frequency of the pulse, but increases its tension. These effects vary according to the quantity of water taken and the rapidity with which it is swallowed; the greater the quantity and the more quickly it is drunk the greater the result.

The effect upon the pulse-rate is probably due to stimulation of the terminal branches of the vagus in the stomach, upon the tendency to stimulation of the vaso-motor centre.

When drunk warm it causes diminished tension and a slight increase of pulse-rate, due, it has been thought, to nausea, causing a lowering of tone in the nerve-centres.

Hot-water drinking (110° to 115° F.) seems to exert much the same influence on pulse-rate and tension as cold water, but slightly raises the general temperature of the body.

The drinking of hot water is indicated when it is desired to stimulate the stomach to increased action and to wash out the system generally. It is more diffusible than cold water, and does not depress either by lowering temperature or by using vital force for the purpose of reheating the organism.

Cold-water drinking sometimes stimulates the lower portions of the alimentary canal. Many invalids, with failure of digestive power, find that warm water taken with or just after food is less depressing than cold water.

For the purpose of washing out the system, aiding the removal of tissue waste and the escape of the various toxines, water should be taken in the early morning, the next best time being one hour before the mid-day or evening meals. Some gouty patients find a tumblerful of hot water at bed-time of great service.

The dosage of water varies. Women, who as a rule take too little fluid, except with food, have to commence with from four to six ounces. The subjects of gastric atony have also to take small doses at first. In other cases, especially in the gouty, a pint of hot water may be taken every morning fasting, the drinking being spread over ten or fifteen minutes. Nausea is sometimes produced by drinking hot water. This may be got over by making the water hotter, or by adding a dessertspoonful of fresh lemon juice to each pint. This addition as a rule suits the gouty and dyspeptic—recent researches showing that it checks the formation of duodenal ferments and toxines.

It has been alleged that the drinking of large quantities of water dilutes the blood unduly, but expert evidence is against this view, and shows that part of the excess of water quickly passes through the kidneys, the rest going to dilute the various fluids in the tissues. It is probable, however, that in marked gastric atony the fluid does not leave the stomach as quickly as it should, and so aggravates the morbid condition. Water-drinking causes marked diuresis, and urea, chlorides and phosphates are passed in increased quantities; the flow of the saliva, pancreatic fluid, and bile is augmented.

External Uses of Water.—The physiological effects are very fully treated of in the article "Balneology," vol. i. p. 428, and the special effects of baths given in water containing various salts and gases are also dealt with in the same article.

The hot bath, 96° to 110°, causes an increased flow of blood to the skin, the pulse is quickened, as is also the respiration. The skin becomes red and perspiration follows.

The tepid bath, 84° to 94°, has a calmative effect, and does not excite either the nervous system or the circulation.

The cold bath, 50° to 60°, at first causes loss of superficial temperature, increased capillary contraction, and an action on the central nervous system through its peripheral branches. Then reaction takes place, followed by redness of skin, increase of temperature, and a certain amount of nervous excitement.

The effects of all these baths depend upon the length of immersion.

Average duration:—hot bath, 8 to 10 minutes; tepid bath, 10 to 60 minutes; cold bath, 3 to 10 minutes.

Longer immersions in either hot or cold baths are often followed by marked depression.

Hydropathic baths and methods consist of thermal and mechanical measures which stimulate the whole body or parts thereof. These give rise to—

(1) Raising or lowering of temperature.
(2) Improved circulation through the peripheral vessels, lymph spaces and channels, and increased elimination of toxines.
(3) More efficient oxidation.
(4) Improvement in general vascular and nervous tone.

HYDROPATHIC METHODS. — The chief measures employed are the following:—

The Shower Bath.—Temp. 100° to 60° or 50°. Duration, one to two minutes.

The Needlebath or *douche en cercle.*—This consists of two curved horizontal pipes connected by a number of smaller vertical pipes. These vertical pipes are perforated with small apertures at intervals of an inch and a half, the result being that many forcible jets of water impinge simultaneously upon practically the whole body surface. Temp. 100° to 60° or 50°. Duration, one to three minutes.

Douches.—(*a*) *Ascending.*—The patient sitting on a seat, like that of a water-closet, water is delivered vertically upwards through a $\frac{3}{4}$-inch nozzle, or through a small rose, so as to impinge upon or even enter the fundament. Temp. 100° to 60° or 50°. Duration, half to two minutes.

(*b*) *Descending.*—A stream of water from a $1\frac{1}{2}$-inch pipe is poured from above. The patient stands under this, moving slightly backwards and forwards to allow the water to pass down back and front of body. Temperature and duration as last bath.

(*c*) *Spinal.*—A stream of water directed from $1\frac{1}{2}$-inch pipe and played up and down the spine. Temperature and duration as last.

(*d*) *Scottish.*—Alternate jets of water at temperature 100° and 50°, played upon the spine, changing six times in the minute. Duration, half to two minutes.

(*e*) *Aix Douche.*—A combination of hot douche with massage. The patient sits on a short-backed wooden chair, the feet being covered with hot water, and a fixed spray at 98° plays upon the back and shoulders. Meanwhile the masseur, directing a stream of water from an open hose-pipe upon the part, rapidly manipulates the front of the body and afterwards the back. The proceeding may be varied by a second attendant simultaneously treating the back. At the end of the bath the patient stands at the extreme end of the room grasping a bar, and a strong pressure douche is played over the body in the direction of the lymph stream, following the course of the lymphatic vessels. A valuable modification, very useful in many cases, is to let the patient lie in a shallow elevated bath, and the attendant giving the massage under the douche, finishing with the final douching as mentioned above.

(*f*) *Underwater Douche.*—An invaluable aid to treatment, especially that by natural mineral water baths. While the patient is sitting in the bath a pipe with a $\frac{3}{4}$-inch nozzle is placed in the bath, and water under pressure at a temperature of from 96° to 110° is applied to the affected parts, the force being graduated by keeping a cushion of water between the end of the nozzle and the part douched.

Packs.—(*a*) *Hot*—general or local. A thick blanket is folded and rolled up in bottle form. Boiling water is poured through the two ends in turn: it is covered with a large hot towel, and as much superfluous moisture as possible wrung out. It is then spread out on the top of two large dry blankets spread on the bed; on a spare one, if the patient is confined to bed. The patient, divested of clothing, is placed on the hot wet blanket and rapidly covered up by this and the dry ones. Hot water tins should be placed outside the blankets and to the feet, and cold water cloths to the head. The operation should be done quickly, so that the wet blanket does not lose heat. Duration, 30 to 60 minutes.

(*b*) *Cold.*—Two large thick towels are wrung out of tepid or cold water. The patient is laid on one and covered by the other, and a single blanket is lightly placed over all. Every ten or fifteen minutes the upper towel should be replaced by one wrung out of cold water; the lower one may be changed every hour, if required. Duration, from 30 minutes to two hours.

(*c*) *Mustard.*—Mustard bran is mixed into a thin paste with boiling water. It is spread on muslin rag and covered by a piece of the same. It is then placed over the affected part and kept on for 10 to 20 minutes, covered by a flannel bandage.

Compress: hot or cold.—A piece of thick Turkish towelling is wrung out of hot or cold water and applied to the affected part. It is then covered by flannel, outside which a piece of Mackintosh sheeting is applied. Duration, 20 to 30 minutes.

HOT AIR APPLICATIONS.—(*a*) *Turkish Bath.*—Fully described in article "Balneology," vol. i. p. 438. Temperature, 115° to 200°. Duration, 20 to 60 minutes.

(*b*) *The Hot Air Box.*—The patient sits in this with head protruding, the feet being placed in hot water and a cold towel applied to the head. Temperature, 108° to 120°. Duration, 8 to 12 minutes.

(*c*) Hot air bath given in bed, patient covered by wicker cage; lamp out of bed and heat conducted through metal tube. Duration, 15 minutes to two hours or more.

(*d*) Local hot dry air applications:—

(1) In box as before described through holes cut for insertion of hands or feet.

(2) "Greville" Method.—The limbs placed in cylinders heated by electricity. Temperature, 180° to 450°, or as much as can be borne. Duration, 30 to 80 minutes.

(3) "Dowsing" Method.—The electric lamp bath with reflectors. Temperature and duration as above.

(4) "Tallerman" Method.—The limb placed in a copper cylinder heated by a large number of gas jets. Temperature, 110° to 250° or more. Duration as above.

The whole body as well as individual limbs may be treated by the "Greville" and "Dowsing" methods.

VAPOUR APPLICATIONS:—

(*a*) Russian Bath.—A chamber heated by steam to a temperature of 110° to 120°. Duration, 10 to 20 minutes.

(*b*) Vapour Box Bath.—Temperature, 108° to 120°. Duration, 8 to 15 minutes.

(*c*) Local Vapour Baths.—Hands or feet placed in box through holes cut for purpose. Temperature, 110° to 120°. Duration, 5 to 20 minutes.

Sitz-baths.—Patient sits in special bath, the water just covering the groin. The back and shoulders should be covered by a blanket, and the feet should also be wrapped up. May be given hot, tepid, or cold. Duration, 2 to 10 minutes.

Hydropathy is of service in both acute and chronic disease.

ACUTE DISEASES.—*Acute Rheumatism.*—A hot blanket pack, as before described, is often most useful. May be repeated twice daily if required.

Rheumatic Hyperpyrexia.—These cases are so quickly fatal that rapid and energetic methods are called for. The writer is strongly in favour of the *immediate* use of the cold bath, and believes that in many cases the time wasted in cold sponging or cold packing has determined a fatal issue.

Whenever the temperature in acute rheumatism touches 105° the patient should be carefully lifted in a sheet by five persons—one at each corner, the fifth supporting the head. A bath should be placed close to the bed, and should contain from 18 inches to 2 feet of water at 92°. This should be cooled down by the addition of ice or cold water to 72°, and the patient kept in till the temperature falls below 100°. Ice should be applied to the head during the bath. Careful attention must be paid to the condition of the heart, which will, of course, govern the duration of the bath: this is usually 10 to 30 minutes. The patient must be carefully lifted in the sheet and replaced in bed on a hot blanket, with a hot water bottle to the feet. After a short time both sheet and blanket may be drawn away.

The bath should be repeated as often as the temperature reaches 105°.

The writer has known as many as eight baths given in twenty-four hours with recovery of patient.

Enteric Fever.—Various opinions are held by competent authorities as to the respective value of cold baths, cooling packs, cooling towels, and cold sponging in this disease; but nearly all are in agreement as to the benefit derived from one or other of these methods.

The advocates of the routine bath treatment say that whenever the temperature rises above 103° the patient should be immersed in a bath at 90°, cooled down to 70° or even 60°. Period of immersion, 10 to 20 minutes; the method of administration being as before described. The objection to this treatment is that it needs to be given with the greatest skill and care, as, when bowel complications are present, much damage may be done by injudicious movement, and preference is often given to the following methods:—

Cold pack, as before described. This method may be continued until the temperature falls below 100°.

Cooling Towels.—Pieces of thick Turkish towelling, 2 feet by 1 foot, are wrung out of tepid water and applied from the top of the chest to lower part of abdomen, across the front of the body, and a piece of flannel placed over them: every few minutes the towels may be wrung out of cold or iced water and changed. This process does not necessitate any movement of the patient, and can be applied continuously.

Sponging with cold or iced water may be done every hour, the various parts of the body being uncovered, sponged, and covered in turn.

Ice packing is sometimes resorted to in extreme cases, but as a rule is not so convenient nor so efficacious as the above methods.

During these applications of cold to the body the head should be kept cool by an ice cap or cold-water bag.

Scarlet Fever.—In those cases of scarlet fever where the rash fails to come out and the patient is threatened with death from hyperpyrexia, the treatment is exactly the same as for rheumatic hyperpyrexia.

The bath and not the milder cooling methods is the only remedy.

Typhus Fever.—Here the cooling pack is beneficial.

Pneumonia.—In the pneumonia and broncho-pneumonia of children the cooling towel is most useful, and should be used whenever the temperature rises above 102°.

In adults the application of cold has to be made more cautiously, but whenever the heart seems to be failing from high temperature the cooling pack or cooling towels should be used; and in extreme cases with very high temperature the cold bath, but the latter should be used with the greatest care. Large enemata of cold water reduce temperature and are sometimes useful.

Uræmia.—The hot blanket pack, before described, is sometimes useful here, but not nearly so much so as the hot air or vapour bath given in bed. The bedclothes being supported by cages, a large spirit lamp is placed on the floor at the foot of the bedstead, and the hot air or vapour is conducted into the bed by means of a tube. The bath may be kept going for from fifteen minutes to two hours, and may be repeated as often as required. It has frequently succeeded when all other methods have failed, especially in nephritis following scarlet fever, and in some cases of puerperal convulsions.

Acute gout is often at once relieved by local hot air or vapour baths.

SUBACUTE AND CHRONIC DISEASES.—*Rheumatism and gout* are benefited by the use of the hot air and vapour baths, general or local. In cases of gout the vapour generally seems more useful, and is an invaluable adjunct to treatment by natural mineral water baths.

Lumbago, myalgia, sciatica, neuralgia, and neuritis are also efficiently treated by these methods, with the help of the hot underwater douche; also by the Aix douche. The more chronic forms of sciatica are relieved by the Scottish douche.

Rheumatoid arthritis, if of the rheumatic or gouty type, is relieved by the local application of hot dry air, especially in conjunction with the constant current or sinusoidal or electric bath; in the more purely neural forms the hot air is of limited service, and should be used with great care, the electric bath being much the most effective treatment.

Neuritis is often benefited by the application of mustard packs over the painful nerves, especially over tender spinal points, and they are often given in connection with the sinusoidal electric baths which have done so much for neuritis.

Affections of the skin, as dry eczema, psoriasis, lichen planus, etc., are well treated by hot dry air; the more inflammatory condition by the steam box bath or local application of vapour.

Chronic Bright's disease, renal insufficiency, suppressed gout, lithæmia, and other forms of auto-intoxication, can be safely and well treated by the vapour or hot air baths.

Neurasthenia.—Needle bath, Scottish douche, mustard pack over the cervical ganglia of the sympathetic.

Dyspepsia and Hepatic Congestion.—Needle bath, Scottish douche, mustard pack over cervical ganglia of sympathetic and over liver and stomach.

Piles.—Ascending douche.

Leucorrhœa, dysmenorrhœa, and other uterine troubles, sitz bath with ascending douche.

Obesity.—Needle bath, spinal douche, Scottish douche, Aix douche.

Constipation.—Scottish douche, cold spinal douche, large enemata of water to wash out colon.

ICE.—May be used internally or externally. It is useful sucked, or swallowed in small pieces, in inflammatory conditions of, or hæmorrhage from mouth, tonsils, throat, or stomach, and in vomiting it also lowers temperature and sometimes stops hiccough; placed in the rectum it lowers temperature.

Externally it has many uses. It is applied to the head in inflammatory and feverish affections: to the neck in acute tonsillitis. Application to the spine and over the epigastrium relieves hiccough, and is often placed over lungs and stomach in hæmorrhage from those organs.

It has been used with much success in the treatment of pericarditis and endocarditis, applied for short periods over the heart; and tachycardia has often been relieved by this application. It relieves acute inflammatory affections of joints, and also some cases of neuritis. Certain obstinate cases of acute or subacute rheumatoid arthritis, especially of the knee, have done well with ice massage, twice daily, for half an hour each time. A flat piece of ice is taken in the hand of the attendant, and gentle rubbing is given. The application of the ice bag to the lower spine is said to stimulate the utero-ovarian organs; while applied over the lower part of the abdomen it stops uterine hæmorrhage; it is sometimes serviceable in the early stage of strangulated hernia, enabling reduction to be made without operation.

Hydrophobia. *See* Rabies.

Hydrothorax. *See* Pleura.

Hymen. *See* Vagina.

Hyperæsthesia. *See* Sensation.

Hyperidrosis. *See* Sweat Glands.

Hyperkeratosis. *See* Ichthyosis.

Hyperpyrexia. *See* Hydropathy, Temperature.

Hypertrophy.

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By hypertrophy is meant an increase in the size of an organ or tissue beyond the ordinary limits, the increase taking place along the lines of physiological growth. It may be defined in the following terms: "Hypertrophy of an organ or tissue expresses that condition where its normal bulk is universally augmented by an additional number or by increased dimension of its tissue elements without deposition of foreign substances" (Hamilton). It may therefore be called a *progressive nutritive change* (as opposed to a *retrogressive nutritive change*—a degeneration or atrophy), and when it deviates to any great extent from the types of embryonic development or physiological growth we have no longer to deal with hypertrophy, but with a tumour. We can distinguish histologically between true hypertrophy, or increase in the size of the individual tissue elements, and false hypertrophy (hyperplasia), or increase in their number. In the case of hypertrophy of an organ, both true hypertrophy and hyperplasia are commonly associated, but since it is only possible to ascertain the existence of the former by careful micrometric measurements, we generally include both, as stated in the definition given above, under the common term hypertrophy.

Hypertrophy depends on a disturbance of the equilibrium between absorption and consumption of nutriment in favour of the former, a state of matters which is normal during the period of growth—physiological growth being in fact the natural type of the progressive nutritive changes of which hypertrophy is one. Since normal growth depends on three factors: (1) Inherent predisposition on the part of the cells; (2) a due supply of nourishment; and (3) the amount of tissue waste—it is to these we must look in seeking to understand hypertrophy. It is impossible to draw any hard and fast line between simple hypertrophy and overgrowth resulting from inflammation on the one hand, or between it and certain forms of pathological new growth on the other. Further, it is impossible to differentiate sharply between normal development and hypertrophy. Neither does our definition exclude congenital hypertrophy, since there is no reason for discriminating between intra-uterine and extra-uterine growth; it must also include such conditions as simple overgrowth of nails and hair. The influence of the nervous system on the occurrence of hypertrophy is indirect, and would appear to be twofold: (1) it stimulates the cells of the tissues to perform their functions, and, as we shall see, functional activity is in most cases necessary in order that the cells may be capable of assimilating nourishment; (2) as the great controller of the blood-flow it determines the amount of nourishment supplied to the various organs.

FACTORS INFLUENCING GROWTH AND HYPERTROPHY.—(1) *Inherent Predisposition on the Part of the Cells.*—This varies in the different organs of the body. In some, *e.g.* the thymus, it ceases early; in others—the nervous tissues—it ceases entirely when normal growth is complete; while in others, again—the muscles, glands, and epithelial structures—it persists during the greater part or the whole of life. Speaking generally, however, it is greatest in early life and diminishes with advancing years.

(2) *Supply of Nourishment.*—Increase in the blood-supply alone will not cause overgrowth of all tissues. The conditions under which it may do so are by no means clear. Experimental section of the cervical sympathetic with resulting vaso-dilatation does not by any means invariably cause increased growth in the hyperæmic area. Hunter, however, produced hypertrophy of the spur of a cock by transplanting it into the highly vascular comb. In the human subject we apparently have an example of hypertrophy due exclusively to increased blood-supply in the great development of the corpus luteum of pregnancy as compared with that of menstruation, and occasionally a somewhat similar corpus luteum occurs in the unimpregnated condition, when there is congestion of the pelvic tissues from some pathological cause. In organs whose function is active—the muscles and glands—no amount of hyperæmia alone will cause an increase beyond normal limits. Inflammatory hyperæmia alone, however prolonged, will never cause hypertrophy of either of these structures, but if associated with excessive functional activity it will readily do so. That is,

the cells of such organs are only capable of assimilating extra nourishment if they are actively functioning. On the other hand, the supporting structures of the body, and also its epithelial coverings, increase in size as the result of long-continued hyperæmia; their capacity for assimilation, therefore, is mainly if not entirely regulated by the amount of nourishment supplied to them.

(3) *Amount of Waste.*—Diminished waste must be accounted a relatively unimportant cause of overgrowth. Nutrition and waste cannot be regarded as mutually dependent: it does not follow that nourishment will be automatically supplied where waste is excessive, or that where waste is diminished nutrition is also lessened. Each of the processes has its own conditions, and therefore waste must be a factor in determining the size of individual organs. An example of pathological enlargement, associated with diminished tissue waste, is said to be afforded by subinvolution of the uterus, but inflammatory changes also play an important part in its production; excessive development of fat in certain cases of obesity would sometimes fall into this group.

Clinical Varieties of Hypertrophy.—Hypertrophy of an organ may be a purely physiological process, as in the case of the growth of the uterus and mammary glands during gestation. In the pregnant uterus there is both hyperplasia and true hypertrophy, the muscular fibres becoming increased in number as well as in size; according to Kölliker they are eleven times longer and four times broader than in the unimpregnated condition. Excessive development of the voluntary muscles, either as the result of athletic exercises or in consequence of certain occupations, can scarcely be looked upon as other than physiological; it is, however, essentially the same as any other functional hypertrophy, *e.g.* of the heart in chronic renal disease, the only difference being in the cause which demands the excessive amount of work. As another instance of physiological, or almost physiological, though not compensatory hypertrophy, senile enlargement of the prostate may be mentioned, since, although a cause of disease, it cannot in itself be invariably considered as morbid. If we accept Hamilton's view that the term hypertrophy should be applied to a general but not to a local enlargement of an organ, as for example when we call a local increase of the gland tissue in the breast an adenoma, we may regard cases of enlarged prostate where the middle lobe is specially affected as allied to tumour growth rather than simple hypertrophies.

From a clinical standpoint we may consider hypertrophy under five heads: 1. Compensatory or functional hypertrophy; 2. Hypertrophy due to repeated irritation; 3. Pathological hypertrophy; 4. Unilateral (congenital) hypertrophy; 5. Giantism. Finally, we have to consider what is meant by pseudo-hypertrophy.

1. *Compensatory or functional hypertrophies* occur in many organs as the result of increased work, if there be also an adequate supply of nourishment. The result of excessive functional activity is increased blood-supply, and if the blood contain proper nourishment hypertrophy will ensue; should nourishment be inadequate the organ will become exhausted and cease to function properly, and will probably waste. Compensatory hypertrophy may occur in *paired organs* where one is destroyed or functionally in abeyance for a prolonged period, in *correlated organs* under similar conditions, in *single glandular organs* where a part of the structure is extensively diseased or atrophied, and in *muscular organs* when a mechanical resistance necessitates an increased output of work. Examples of compensatory hypertrophy in paired organs are seen in cases of congenital

absence of one kidney, and in cases of removal or long-standing disease of one of the organs. In cases of congenital absence of a kidney the remaining organ may be double its normal size, and when the loss takes place in early life it may attain almost equal dimensions; compensation is less complete after growth has ceased. The enlargement is due to true hypertrophy and hyperplasia of the kidney elements; an increase in the number of the glomeruli, however, occurs only in the case of congenital absence of one organ. An analogous compensatory hypertrophy has been observed in the lungs, suprarenal capsules, testicles, and other paired organs. A functional hypertrophy of the kidneys is sometimes seen in cases of polyuria. Thoma points out that compensatory hypertrophy may occur in paired organs where increased function cannot be assumed by the remaining organ, *e.g.* after excision of one testicle in a newly-born animal. In such cases the hypertrophy may be supposed to depend on nervous influence. It must also be stated that, according to one view, enlargement of a congenitally single kidney is to be looked upon as due to fusion of the two structures. It is also said that the enlargement of the active kidney, when the other is removed or extensively diseased, is due mainly to increase in the lymphatics and dilatation of the blood-vessels. Compensatory hypertrophy of correlated organs is seen in the glandular enlargement which occurs after splenectomy, and also in the enlargement of the pituitary body which has been found after experimental removal of the thyroid. In cases of pressure atrophy of part of the liver (as from tight-lacing) compensatory hypertrophy of the remainder may be observed. Similarly we have enlargement of the part of the thyroid left behind after partial thyroidectomy.

From a clinical point of view the most important group of functional hypertrophies are those found in hollow muscular organs. Any long-continued rise in the blood-pressure will cause thickening of the muscular coats of the arteries and cardiac hypertrophy. The individual fibres of the heart muscle become enlarged, and there is as well a numerical increase. A similar change occurs when the pressure in any chamber of the heart is raised, *e.g.* as the result of valvular disease or obstruction to the pulmonary circulation. Cardiac hypertrophy is, in fact, one of the best examples of the importance of some of the factors we have enumerated. It arises most quickly in young persons, it is seen in greatest perfection in aortic incompetence and Bright's disease, where the work of the heart is enormously increased, and we too often have opportunities of observing its failure when an insufficient quantity of nourishment is supplied, as where the coronary arteries are thickened and partially obliterated.

Another important example of functional hypertrophy is that occurring in the wall of the bowel above the seat of a chronic obstruction of the lumen. This shows itself by the presence of visible peristaltic waves, and by observing the direction in which these pass, and the pattern to which they give rise, we can often form a fairly exact notion of the seat of the obstruction. It is unnecessary to multiply examples of such compensatory hypertrophy; we find it in the stomach in cases of pyloric stenosis, in the bladder in cases of urethral stricture, etc.

Functional hypertrophy of voluntary muscles has already been mentioned. A similar condition may arise from disease. Thus true muscular hypertrophy is met with in Thomsen's disease, where there is increased tonicity of all voluntary muscles; it also occurs in some cases of infantile cerebral paralysis from a like cause. The rare condition described by Gowers as muscular hypertrophy, which is associated with diminished power and proneness to exhaustion, without alteration in the mechanical or electrical irritability, may also be mentioned. It would appear to be more closely related to the pathological hypertrophies than to the functional type.

2. *Hypertrophy from Irritation.*—Many instances of this will at once suggest themselves. As examples of overgrowth due to recurring pressure or friction, corns, various trade callosities, and milk spots on the pericardium may be cited. Chronic inflammation often results in a hypertrophy of the connective elements in its neighbourhood, as seen in the thickening of the pleura and periosteum after pleurisy and periostitis; the induration of the skin met with in chronic eczema has a similar origin. For the immediate cause of the hypertrophy in such cases we must look to the hyperæmia and consequent increase of nourishment caused by the irritant. We have already seen that while hyperæmia will not cause overgrowth of glandular or muscular organs unless at the same time there is increase of function, that is, unless the cells are stimulated to utilise the increase of nourishment, the cells of the supporting structures of the body, whose function is a passive one, are more directly dependent for their growth on the amount of nutriment supplied to them.

3. *Pathological Hypertrophies.*—In the above-mentioned instances the hypertrophy has been, if not physiological, yet in the main beneficial, and its proximate causes, at least, have been fairly recognisable. But we have also to deal with a series of cases in which the hypertrophy is usually harmful, and in which the causes are by no means clear. Thus senile enlargement of the prostate stands on the borderland of the physiological and pathological; it is a common sequel of advancing years, and in some cases it approximates to a tumour, yet, unless it give rise to urinary symptoms, it can scarcely be looked on as a disease. The enlarged thyroid of Graves' disease, the hypertrophy of the pituitary body found in some cases of acromegaly, the enlargement of the spleen found in some cases of anæmia, congenital syphilis, and rickets, the adenopathies of Hodgkin's disease, the hypertrophied thymus found in some infants, which may at this age be the cause of sudden death from suffocation, the hypertrophic changes in the bones accompanying certain chronic lung diseases, and the exostoses of the facial bones in leontiasis ossea, are a few examples of this group. Some of them appear to be primary maladies, others to be the results of the associated disease, while in other cases they are more closely allied to tumour growths.

4. *Congenital hypertrophy*—or, as it is more correctly termed, *hemihypertrophy*—is a rather rare condition. In its most typical form it consists of a total hypertrophy of one-half of the body involving the head, trunk, and extremities, the asymmetry being complete in every respect, even extending to the ears, tonsils, two halves of the tongue, and external genital organs. The differences in size may best be illustrated by a few measurements of a case observed by the writer. Ears, 4·5 and 4 cm. long; arms, 18·5 and 17 cm. in length, 15·5 and 13·5 cm. in girth; legs, 25·5 and 22 cm. in length, 24·5 and 17 cm. in girth; mid-sternum to spine at level of nipples, 20·5 and 17·5 cm. The hypertrophy involves mainly the soft parts—the skin, muscles, and connective tissues; the bones themselves, however, are affected in many cases, especially where the length of the limbs is different on the two sides. The overgrowth is as a rule most evident in the more distal parts of the limbs—in the hands and fingers, and feet and toes. The skin is sometimes more active on the hypertrophied side, the hair is thicker and more abundant, the nails are coarser and grow more quickly, and the eruption of the teeth is earlier. The temperature is often, though not always, a degree or two higher on the affected side, and the hypertrophied parts are more susceptible to heat and

cold. As a rule the muscular power is greater on the side of the hypertrophy. There are no nervous symptoms, and the internal organs are normal. There remains only one prominent feature of such cases to be mentioned: the presence of nævi and other cutaneous vascular anomalies. These exist in a large proportion of the cases, sometimes in the form of simple capillary nævi, occasionally of larger telangiectases. At times the skin appears normal while the patient is at rest, but when he cries or is excited large patches of cutaneous congestion, differing from nævi only in their temporary duration, are produced. These vascular abnormalities are never strictly unilateral, but are more or less generally scattered over the body. Another not infrequent appearance is a condition of marbling of the skin like that seen in pseudo-hypertrophic paralysis. True congenital hypertrophy is always stationary, the relative size of the parts "being preserved throughout life."

Not all cases, however, conform to the description we have just given. Hemihypertrophy is much more commonly limited to one limb, or even to a single digit, than widely spread in its distribution. A large number of cases have been reported where hand and foot alone have been involved. We may regard all these—simple non-progressive hypertrophies, usually strictly unilateral, with preservation of the normal appearance of the parts, without the occurrence of tumours or other deformities, the skin often being the seat of vascular anomalies—as belonging to the group of "true congenital hemihypertrophies."

In another group of cases—"false hypertrophies"—a somewhat different condition obtains. In them the abnormality is usually more limited in extent, but greater in degree. Thus we may have hypertrophy of a hand associated with giant growth and deformity of a finger, with syndactylus, or with tumour growth, usually either lipomatous, or of the nature of an enchondroma. Again, the hypertrophy may lead to ankylosis of the joints, or it may be combined with elephantiasis of the limb. These so-called false hypertrophies also differ from the preceding in not being invariably unilateral, congenital, and stationary; sometimes both feet, or a foot and hand on opposite sides, are affected; the hypertrophy may not develop until childhood or adolescence, it may progress with varying rapidity, with periods of quiescence. Sometimes amputation of a giant finger has been followed by overgrowth of the arm. In fact, false hypertrophies are more closely allied to tumour growth than those just described, and are commonly associated with some other developmental anomaly. Still, the two conditions must not be too sharply differentiated; in true hypertrophy we not infrequently find that one or two fingers or toes have not the same proportionate development as the rest; they may be unduly large or of natural size. In such a case the corresponding digits of the opposite side are sometimes also abnormal.

Greig (6) has proposed an anatomical classification of cases of unilateral hypertrophy. He divides cases into those where the affection is limited to the head and face, and those where it is not so limited. Each group is further subdivided into cases in which the bones only are involved, soft parts only involved, and both bones and soft parts involved. Unilateral hypertrophy limited to the head and face deserves some further notice, and although it is not always congenital may be most fittingly considered here.

Unilateral hyperostosis, or hemihypertrophy limited to the bones of the face, must not be confounded with leontiasis ossea. Greig found that the age of onset varied from three to thirty-five years, the average being fourteen. Four out of fourteen cases collected by him were mentally defective, one was epileptic, and in one there was strabismus. The hyperostosis is sometimes accurately limited to the region supplied by the trigeminal. Hemihypertrophy limited to the soft parts, as well as that in which both soft parts and bones are involved, is usually congenital. In such cases the cheeks and lips are often the places most affected, and the teeth and hair may participate in the overgrowth.

It might be supposed that the distinction between hemihypertrophy and hemiatrophy would not always be easy, but in practice this difficulty does not arise, the hypertrophied side always appearing so abnormally large as to prevent the possibility of error. It is said, however, that hemihypertrophy is sometimes associated with hemiatrophy of the opposite side. Hemihypertrophy must not be confused with the slight degree of asymmetry which is so commonly seen in normal individuals.

The pathological changes in these cases are little known; there seems, however, to be both true hypertrophy and hyperplasia, mainly of the connective tissue elements. No abnormality of the vascular or nervous systems, except thickening of the arterial walls, has been described. The viscera are normal. The causes of the condition are unknown. Males are more frequently affected than females, and the right side more often than the left. There does not appear to be any hereditary tendency to this, or indeed to any other congenital deformity, in the families of those affected. It seems most likely that it is due to a developmental error in the middle lamina of the blastodermic membrane, but the following theories have also been advanced:—(1) Congenital lesion of the vaso-motor centres, with venous stasis. (2) Partial intra-uterine strangulation of the affected limbs. (3) Congenital defects in the lymphatic system. (4) Fusion of twins. (5) Nervous influences. The distribution of hyperostosis of the face and cranium over the area supplied by the fifth nerve has been alluded to. Greig, considering the comparative frequency with which mental defects are associated with facial hemihypertrophy, especially where the bones alone are involved, believes that the condition is due to an intra-uterine meningitis or cerebritis, which irritates the nerves while their function is yet entirely trophic. He would explain all forms of hemihypertrophy on this hypothesis.

5. *Giantism.*—As is the case in the opposite condition—dwarfism—the nature and causes of giantism are obscure, and its various forms are not well known. Much of the writing on the subject dates from the seventeenth and eighteenth centuries, and, however great its antiquarian interest, is of little scientific value. Probably the largest authentic giants are Frederick the Great's Scottish giant (8 ft. 5 in.), the Irish giant, whose skeleton measures 8 ft. 6 in., and the "Elsässer Bauer," who lacked only half an inch of being nine feet. The often recurring accounts of men whose stature was ten, twelve, or even fifteen feet, must be relegated to the domain of myth.

Of late years, and especially since the recognition of acromegaly as a well-defined disease, which is not infrequently associated with giantism, renewed attention has been directed to the subject. Giantism may be congenital, or it may occur during youth or adult life. Congenital giants are rare, but every now and then children weighing fifteen to twenty pounds come into the world. In such cases the growth during the first year or two is often rapid, and a precocious puberty is reached at five or six years. Thereafter there is premature ageing, the growth is slowed, and the subjects generally die early.

Giantism may occur in later life under a variety of circumstances, and associated with several other diseases, of which the most common is acromegaly. Sternberg (8), to whose writings we are largely indebted, divides giants into the following groups:—*A*. Normal giants, without any deformity, who enjoy good health, and reach old age. *B*. Pathological giants. (i.) Associated with acromegaly. (ii.) With multiple tumour-like exostoses, leontiasis ossea, or facial hyperostosis. These often show signs of pressure on the brain or cranial nerves. (iii.) Combined with facial hemi-hypertrophy (unilateral hyperostosis). (iv.) With multiple curvature of bones, such as scoliosis, bending of the arm bones, genu valgum, etc.—probably osteitis deformans of Paget. (v.) Giantism has also been seen associated with congenital syphilis, tumours of the testicle, and after castration (?).

Since many of the reported cases of acromegaly have occurred in giants, and many museum skeletons of giants, *e.g.* the Irish giant, have been found to show signs of acromegaly, it appears that something more than a merely accidental relationship must subsist between them. Langer, before the recognition of acromegaly, divided cases of giantism into those with normal skulls, and those who had an enlargement of the hypophysis. The latter he called pathological, and many of them are now known to have been the subjects of acromegaly. It has been said that all giants are acromegalians, but the incorrectness of such a statement is obvious from the above grouping of the cases. Another view of the relation between the two conditions is this: that when the disease occurs in youth, giantism results; when in later life, acromegaly. We now know, however, that typical acromegaly may develop in young persons. It appears that while about 40 per cent of all giants suffer from acromegaly, only about 20 per cent of all acromegalians are giants—that is, acromegaly is more common in giants than *vice versa*. As Sternberg's classification shows, we may have along with giantism not only acromegaly, but a variety of other more or less rare diseases of the bones—leontiasis ossea, osteitis deformans, etc., and it has been argued that giant growth may predispose to the occurrence of such widely-spread dystrophies and lesions of the supporting structures of the body. Much further observation is, however, required before the relations of giantism to acromegaly and these other diseases can be known.

PSEUDO-HYPERTROPHY.—In pseudo-hypertrophy of muscles there is not merely a replacement of atrophied muscle-fibres by fat, but there is actual hypertrophy of the fat and connective tissue. The affected muscle-fibres are abnormally small; most of them, however, preserve their normal striation, but where the change has progressed farthest they show granular degeneration and other changes. The condition is a primary disease of the muscles due to an inherited tendency.

TREATMENT.—Little need be said on this point. Since compensatory hypertrophy is in the main beneficial, and is, in fact, what we aim at in dealing with the conditions to which it is due, we have to do our best to further it in most cases. The indications are furnished by our knowledge of its proximate causes. We endeavour to supply appropriate nourishment, to adapt the stimulus to the capability of the hypertrophying structure—as for example when we order rest in a case of cardiac valvular disease in order to give the ventricular walls time to gain strength and cope with the effects of the lesion, and finally (though this is less often within our power) to diminish tissue waste. Apart from this, compensatory hypertrophy, as such, requires little or no treatment. In the case of the morbid conditions grouped under the heads of pathological and inflammatory hypertrophies, the treatment—further than if possible removing the cause—will vary according to the organ affected. Reference must be made to the special articles for particulars. Hemihypertrophy does not admit of much treatment.

Benefit is said to have followed the application to the limbs of elastic pressure so as to retard growth. In some cases surgical measures may be needed.

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Hypnotics.—A true hypnotic is a remedy which, when given in quantity sufficient to induce sleep, does so without disturbing the relationship of the mental functions to the external world; a narcotic not only induces sleep, but profoundly disturbs this important relationship. The *rôle* of these remedies should be carefully differentiated, and it is well to recognise the fact that the injudicious use of the former exerts a distinct influence towards the indulgence of the latter more dangerous remedy. If this were more thoroughly appreciated, we would meet with fewer cases of morphinomania, chloral and cocaine habit than unfortunately pertain at present. For the induction of sleep the following points may be necessary:—

I. To produce a slight degree of anæmia of the cerebrum, *e.g.* by dilating blood-vessels in other parts of the body, or by stilling any excited cardiac action.

II. To lessen the functional activity of the organism as a whole, and especially in certain cases, *e.g.* that induced by derangement of the digestive tract (as by a too heavy late supper, or a dinner indulged in not wisely but too well), and also that induced by pain or discomfort in some peripheral area. A consideration of these points shows us that a hypnotic may act in one of two ways:—

1. By an indirect action in removing the cause that is preventing sleep.
2. By a direct action—that of a paralysing action on the cerebrum.

Most of the hypnotics in common use belong to the latter class; some of these, *e.g.* the bromides, have a slight action both as direct and indirect hypnotics. The use of hypnotic substances is greatly abused, and for this state of affairs medical men are in part responsible. Not by any means infrequently do we meet with patients complaining of more or less minor ailments, whose history reveals them to have been the slaves of sulphonal or chloral hydrate, taken indiscriminately for many months or even longer, and the treatment of such cases is much more difficult in virtue of that habit. It is a wise rule to observe that in a considerable proportion of those cases in which the administration of a hypnotic is really called for, one should be chosen which there is little prospect of the patient taking a particular fancy to. Herein paraldehyde is valuable. But we must bear in mind that with no class of medicinal remedy is there a greater tendency to idiosyncrasies on the part of patients, and these idiosyncrasies are usually of a nature calling for special vigilance on the part of the practitioner. It may be that the dose given was insufficient, and in place of a paralysing action in the cerebrum, the very reverse action was induced, to the detriment both of the patient and practitioner. Again, the dose may have been excessive, and a resulting depression of the heart and vital powers causes additional anxiety.

Special care must be taken with the use of all recent remedies with advertised powerful hypnotic action, said to be unaccompanied by any such untoward effects. A few general rules can only be given; no hard and fast rules can be formulated.

1. Ascertain the cause of the sleeplessness, and if, as is very frequently the case, that depends on a general state of health, calling for a short or long complete rest with change of air, scenery, occupation, etc., that must be enjoined, and to the exclusion of all so-called hypnotic remedies.

2. General tonic remedies, and it may be special tonics, such as digitalis, are very frequently the line of treatment indicated.

3. In cases of brain unrest and irritability resulting from brain fatigue, in addition to enjoining the necessary brain rest, a judicious course of bromides will be found most appropriate. The following prescription can be cordially recommended:—

℞	Sodii bromidi	℥ss.
	Quin. hydrobrom.	ʒss.
	Tinct. aurantii	℥j.
	Aquæ ad	℥vj.

A tablespoonful to be taken in a wineglassful of water an hour before bed-time. For use under medical directions only.

4. In other cases it may be advisable to fall back on sulphonal, bromidin, chloretone, trional, and the like—in the use of which two points should always be kept in mind:—

(*a*) The remedy should be prescribed for a stated time, and only taken under medical supervision.

(*b*) The dose administered should be carefully selected to meet the requirements of each case; the great variations in susceptibility of different patients must ever be kept in mind.

Hypnotism.

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INTRODUCTION.—Hypnotism, although officially recognised, can hardly be described as well known. Hence facts must be cited and explanations given which would be out of place in dealing with more familiar departments of medicine. For those who wish to understand, and possibly to practise, hypnotism, something more is required than a mere account of cases which have been relieved or cured by it. Information on many points is necessary, as the questions put to me show, and to these the following article is an attempted reply.

HISTORICAL

Just as chemistry arose from alchemy, astronomy from astrology, so hypnotism had its origin in mesmerism. Hence, among hypnotic phenomena are to be found those mesmeric ones which have stood the test of rigorous investigation, and are now explained in a more scientific way. Phenomena such as Mesmer (*b.* 1734, *d.* 1815) described had been observed from an early date in human history, but after his day they were usually called by his name, and explained by his theory, *i.e.* by the action of a mysterious force or fluid which emanated from the operator.

In 1837 a great mesmeric revival began in England, of which John

Elliotson was the prime mover. After seeing the experiments of Dupotet, Elliotson introduced mesmerism into his hospital practice and teaching. Shortly afterwards, however, this was forbidden by the hospital authorities, whereupon he resigned his appointments and continued his work independently. Mesmeric infirmaries were opened in London, Edinburgh, Dublin, and elsewhere, thousands of patients treated, and hundreds of painless operations, including amputations of the thigh, etc., performed.

Elliotson's influence spread to India. There James Esdaile, surgeon to the East India Company, heard of his work, and, in 1845, tried to mesmerise a Hindoo convict about to undergo a surgical operation. The result was successful, and, at the end of a year, Esdaile reported 102 painless operations to the Government. Shortly afterwards he was appointed to a Government hospital in Calcutta, so that he might further test mesmerism as an anæsthetic. Until he left India, in 1851, Esdaile occupied similar posts, and recorded nearly 300 painless major operations and many thousand minor ones.

The mesmeric work of Elliotson and Esdaile was bitterly opposed by the medical profession, and the movement practically ceased at Elliotson's death.

James Braid (born 1796, died 1860) commenced his mesmeric investigations in November 1841, after seeing Lafontaine produce apparently genuine phenomena. Braid demonstrated the subjective origin of mesmeric phenomena, substituted the term hypnotism for that of mesmerism, invented the terminology which we still use, and employed hypnotism with good therapeutic results. He published many works on the subject, all now out of print, and most of them little known. A list of those which I have been able to trace, thirty-eight in all, is to be found in the *Revue de l'Hypnotisme* for June 1898.

In 1859, Azam of Bordeaux became acquainted with Braid's work, and commenced to investigate the subject. About the same time, Broca and Velpeau drew the attention of the Académie des Sciences to Braid's researches. The interest thus excited soon faded, and it was not until 1883 that *Neurypnology*—Braid's first published book—was translated by Jules Simon. Despite the fact that Braid's theories were adopted by such well-known men of science as Professors Carpenter and John Hughes Bennett, the practice of hypnotism apparently ceased in England at his death.

In 1860, Liébeault began to investigate mesmerism, and, four years later, started his gratuitous practice of hypnotism amongst the poor of Nancy. He published *Du sommeil et des états analogues considérés surtout au point de vue de l'action de la morale sur le physique* in 1866, but of this one copy alone was sold, and Liébeault's work was ignored by the profession until Bernheim became a convert in 1882, and employed hypnotism in his hospital practice. In 1884, Bernheim published *De la suggestion dans l'état hypnotique et dans l'état de veille*, and, in 1886, *La thérapeutique suggestive.* Liébeault's name now became well known, and doctors flocked from all countries to study his methods.

Independently of Liébeault, Charles Richet, in 1875, drew attention to the genuine nature of hypnosis, and termed the condition *somnambulisme provoqué.* In 1878, Charcot began to lecture on the hypnotic phenomena observed in hystero-epileptics. In Germany, Heidenhain and others commenced to investigate the subject in 1880, but the movement they originated soon ceased. Charcot and his followers formed the school of the Salpêtrière; their views, however, are now generally discredited, and have done little or nothing to further the therapeutic use of hypnotism. On the other hand, the movement originated by Liébeault has now spread over Europe, its development in Germany being mainly due to Forel. In England, although Braid was never completely forgotten, the hypnotic revival, apart from my own share in it, arose from Liébeault's influence, and hypnotism, despite some opposition, has now gained a recognised place.

At the Birmingham meeting of the British Medical Association in 1890, a committee appointed to investigate hypnotism reported unanimously, two years later, that they had satisfied themselves as to the genuine nature of hypnotic phenomena, and the value of hypnotism as a therapeutic agent. Hypnotism has now definitely taken its place in medicine and science. No work on general therapeutics is complete without reference to it, and the physiologist or psychologist who ignores its existence, or denies its phenomena, imperils his reputation.

Methods of Inducing and Terminating the Hypnotic State

Inducing Hypnosis.—The chief methods of inducing hypnosis are—(1) Sensory stimulation, *e.g.* passes with contact, fixed gazing, etc.; (2) Central stimulation by suggested ideas. The first was the only one consciously employed by the mesmerists; the latter is the avowed method of the Nancy school. The modern hypnotiser, however, borrows in technique from Mesmer and Liébeault with equal impartiality. The members of the Nancy school, while asserting that everything is due to suggestion, do not hesitate to use physical means, and Mesmer's passes are almost exactly reproduced by Wetterstrand. Fixed gazing generally precedes or accompanies suggestion, and, when these fail, Bernheim has recourse to narcotics. At first Braid relied mainly on fixed gazing; later, he hypnotised as readily in the dark as in the light, and easily succeeded with the blind. As the result of this, he concluded that the influence acted directly on the mind—not through the optic nerves—and that the best method was *direct verbal suggestion.* While it is doubtful whether physical methods alone have ever succeeded, hypnosis can certainly be induced when these methods are excluded. Here, the subject is made to understand the condition the operator wishes to induce, and to expect its appearance. This condition is kept constantly before the subject's mind by the operator's reiterated verbal description of it; nothing more is necessary. Once hypnosis has appeared, it can be reinduced at any time in response to any signal previously arranged by suggestion.

Terminating Hypnosis.—No matter how profound the hypnosis, the subject can always be aroused with ease. It is only necessary to suggest during hypnosis that the condition shall terminate in response to some given signal, such as the audible counting of "one," "two," "three" by the operator. Afterwards the subject invariably responds at once whenever the signal is given.

Experimental Phenomena of Hypnosis

These vary with the depth of the hypnosis, the personality of the subject, and the methods of the operator. In the earlier stages we do not find all the phenomena which characterise the deeper ones, but in the latter we can induce not only its own phenomena, but those of all lighter stages. For convenience sake, therefore, I propose to describe the phenomena which have been evoked in the most profound form of hypnosis, *i.e.* somnambulism.

Changes in the Voluntary Muscles.—The following muscular phenomena have been observed:—*Catalepsy.*—In extreme cases there is general tonic contraction of the muscles. *Flexibilitas Cerea.*—Here, the limbs can be bent like soft wax and maintain any position in which they are placed. *Increase of Muscular Power.*—Sometimes the subjects are able to perform physical feats far beyond their normal strength. *Paralysis.*—The different voluntary muscles may be paralysed singly or in groups. In some cases, the muscles necessary for the performance of a given movement do not contract; in others, the muscles act, but are overpowered by the violent contraction of the antagonistic ones. There are, however, few, if any, changes in the voluntary muscles which are absolutely characteristic of hypnosis, and none which arise independently of direct or indirect suggestion. The most noticeable phenomenon is the long maintenance of an uncomfortable posture, associated with extreme muscular rigidity, without subsequent fatigue.

CHANGES IN THE INVOLUNTARY MUSCULAR AND VASO-MOTOR SYSTEMS.—*Pulse.*—In many instances the frequency of the pulse can be easily altered by suggestion, this being accompanied by corresponding sphygmographic changes. Thus, increased speed is associated with decreased tension, and *vice versa.*

Bourru and Burot, Mabille, Jules Voisin, Artigalas, Rémond, Hulst, etc., report cases of *bleeding* from the skin; Forel, Schrenck-Notzing, Rybalkin, etc., *local redness of the skin;* and Focachon, Rybalkin, Krafft-Ebing, Hulst, etc., *blisters* produced by suggestion. Krafft-Ebing and others cite cases of *alterations of temperature.*

The evidence as to *local redness of the skin* and *bleeding* is convincing in some instances and unsatisfactory in others. As to the alleged *changes of temperature,* further and more careful observations are necessary, while the evidence as to *blistering* is not conclusive.

Forel and other observers have arrested and excited *menstruation* during hypnosis; caused its appearance at a given date by post-hypnotic suggestion, and regulated its duration and intensity. The action of the *bowels* can be easily excited by suggestion. In short, many cases are cited which tend to show that *secretion* and *excretion* are largely under the influence of suggestion.

CHANGES IN THE SPECIAL SENSES, COMMON SENSATION, MUSCULAR SENSE AND APPETITE.—*Vision,* both distant and near, can be altered by suggestion. In some instances, at all events, this is due to changes in the muscles of accommodation. Many instances of suggested hyperæsthesia of the sense of *smell* are recorded. An increase of *muscular sense* is shown by greater power of discrimination between minute differences in weight, and in other ways. *Common cutaneous sensibility* can be increased, the subjects sometimes distinguishing the points of the compass at half the normal distance. *Thermo-sensibility* can also be rendered more acute. *Hunger* and *thirst* can be excited, or temporarily arrested, by suggestion.

The activity of the *special senses* can be decreased or arrested. A psychical *dumbness, blindness,* or *deafness* can be produced, while, in deep hypnosis, *analgesia* and *anæsthesia* can be easily evoked, either simultaneously or separately. Cases in which impressions, which otherwise would have aroused consciousness, are arrested before they reach the higher cerebral centres are usually classed as *negative hallucinations.* The term *positive hallucination* is applied to cases where the memory of past sensory impressions has been aroused by suggestion, and the subject led to believe in their reality.

AUTOMATIC WRITING.—Problems, suggested to the subject during hypnosis, may be recorded after waking by automatic writing. Here, the primary waking consciousness retains no recollection of the hypnotic suggestion. It does not know that the secondary consciousness, after the hypnotic state has been terminated, first solves the problem and then directs the motor acts which record it. It is also unconscious of the motor acts themselves.

HYPNOTIC AND POST-HYPNOTIC APPRECIATION OF TIME.—Many observers have noticed an increased appreciation of time during hypnosis and in the fulfilment of post-hypnotic suggestions. One of my subjects, if told to do something at the expiration of a complicated number of minutes from a given hour, as, for example, 20,845, almost invariably carried out the suggestion with absolute precision, while no recollection of the command or its execution existed in the primary consciousness. Further, the calculations involved were far beyond the subject's normal arithmetical powers, while her waking self possessed no special power of time appreciation.

RAPPORT.—The mesmerists of Elliotson's day believed that a peculiar *rapport* existed between the operator and his subjects. The latter were supposed to be in relation by their senses with those who mesmerised them, but with them alone. Braid pointed out that the condition was a purely artificial one, the result of training, and that, even when it existed, the *rapport* was more apparent than real, as outsiders were able to get into relationship with the subject, although they had to employ special devices for this purpose. Moll's later observations confirm Braid's views.

MEMORY.—In slight hypnosis there is absolutely no alteration in memory, but in the most profound state—somnambulism—there is post-hypnotic amnesia, *i.e.* the subject during normal life remembers nothing of what had taken place in hypnosis. On being rehypnotised, however, he can recall all the events of previous hypnoses, those of normal life he had previously been able to remember, and frequently also many things which the waking self had forgotten.

VOLITION.—I have never, either in this country or abroad, seen a hypnotic suggestion carried out which involved anything opposed to the patient's prejudices, feelings, or moral sense. Bernheim and others base the possibility of hypnotic crime on the fact that hypnotic subjects will sometimes execute imaginary ones, *e.g.* put a lump of sugar into a friend's teacup, when told this is arsenic. Questioning in subsequent hypnosis, however, always reveals the fact that the subject knew exactly what he was doing, and fully recognised the experimental nature of the transaction.

SELF-HYPNOSIS.—Once genuine hypnosis has been induced, it is an easy matter to teach the subject to evoke the condition for himself. Afterwards, he can make the necessary suggestions to himself while awake, and reproduce the state at a given signal. Some of my former patients still employ hypnotism in this way for the relief of pain, the production of sleep, etc.

THE THERAPEUTIC USES OF HYPNOTISM

With the space at my disposal, it is only possible to give a short outline of the therapeutic uses of hypnotism, while illustrative cases must necessarily be few in number and lacking in detail. It is in functional nervous disorders that hypnotism yields its best results. Thus, Bérillon and many others assert that it is specially indicated in the following hysterical affections:—

I. *The convulsive attacks of "grande hystéric" and the symptoms which sometimes persist after them—paralysis, contracture, anæsthesia, etc.*

In this country I have only seen three cases of "grande hystérie" of the true Salpêtrière type. The first, treated by drugs alone, died from cerebral hæmorrhage following a convulsive attack. The second, hypnotised after repeated failures, recovered completely and has remained ten years without relapse. The third, after varied treatment, passed four months in the National Hospital, Queen's Square, where subcutaneons injections of hyoscine, and large and frequently repeated doses of chloral, bromide, etc. were administered, her condition growing distinctly worse meanwhile. On 14th June 1900, when I first saw her, she was unable to walk; there was a contracture of the muscles of the left leg, constant clonic muscular spasm of the muscles of the head, trunk, and extremities on the left side, together with frequent and violent generalised convulsive attacks of the true Salpêtrière type. On June 18th, she was discharged from the hospital and came under my care; she commenced to improve almost immediately under hypnotic treatment, and three weeks later all morbid symptoms had disappeared.

Numerous cases of "grande hystérie," with good results, are reported by Continental observers, and Bérillon, who for three years watched Dumont-pallier's cases at the Pitié Hospital, states that most of the patients made a complete recovery and are now in good health—some married and mothers of families, others occupying responsible business positions.

Voisin reported a case of long-standing hystero-epilepsy with maniacal attacks; the patient recovered and became wardrobe maid at the Salpêtrière.

II. *Monosymptomatic hysteria—monoplegia, mutism, aphonia, hiccough, cough, blepharospasm, discromatopsy, rhythmical chorea, etc.*

The following are illustrative cases from my own practice:—

Mrs. ——, aged 49; singultus of three years' duration, supposed to be due to large uterine fibroid. No improvement from careful drugging, electrical treatment, etc. Hypnotised. Recovered. No relapse after five years.

Miss ——, aged 20; aphonia of four years' duration, with loss of reflex sounds. Paresis of lower extremities. Rapid recovery under hypnotic treatment.

Miss ——, aged 19; violent muscular tremor of extremities of fifteen months' duration. Careful hospital treatment without benefit. Hypnotised. Rapid and complete recovery. No relapse after five years.

Bérillon, de Jong, Wetterstrand, van Eeden, van Renterghem, and many other Continental observers, report successful cases of hysterical aphonia, hiccough, cough, mutism, blepharospasm, chorea, etc.

Dr. A. Gros d'Apt reports a case of hysterical paralysis of the lower extremities, of three years' duration, which was rapidly cured by suggestion.

Van Renterghem records a case of long-standing clonic spasm of the cervical muscles in a man, aged 42, who had been treated without success by drugs, electricity, hydropathy, nerve-stretching, etc., but completely recovered under hypnotic treatment.

A similar successful case is reported by Tatzel. The patient, a man, aged 44, had been compelled to abandon work and, after having spent all his money on medical and surgical treatment, ended in the workhouse. Tatzel also reports two successful cases of paramyoclonus multiplex, while Lemoine cites one of hysterical tremor of twenty-one years' duration, simulating paralysis agitans, which recovered after six days' hypnotic treatment.

III. *The various manifestations of ordinary hysteria—insomnia, dyspepsia, visceral and menstrual troubles, neuralgia, etc.*

The following are illustrative cases from my own practice :—

Miss ——, aged 20; insomnia from infancy, with spinal neuralgia of ten years' duration. Complete recovery after short hypnotic treatment. No relapse after five years.

Mrs. ——, aged 41; migraine, dysmenorrhœa, dyspareunia, chronic constipation, dyspepsia, insomnia, constant depression, spinal neuralgia, etc. Slight hypermetropia, over-corrected by ciliary spasm, causing virtual myopia—could only read third line of Snellen's unaided. All the morbid symptoms, including the ciliary spasm, disappeared after a short hypnotic treatment, and the patient was able to read the bottom line of Snellen's unaided. The ophthalmic observations were made by the late Mr. Bendelack Hewetson. Two years later there had been neither relapse nor return of the ciliary spasm.

Miss ——, aged 28; pain in hip and leg of five years' duration, with inability to walk. Treatment: drugging, rest on back for two months; Weir-Mitchell; massage; electricity; hydropathy; Paquelin's cautery to leg — about 20,000 applications in all. Meanwhile, the patient grew steadily worse; all treatment was then abandoned, and she was considered incurable. Complete recovery followed two days' hypnotic treatment; the patient exchanged her bath chair for a bicycle, and now, four years later, is well, active, and strong.

As a large proportion of my patients suffered from hysteria, I could cite many cases similar to those just given. In all the type was identical, the details alone differed. One or more of the bodily functions was performed consciously and painfully, and some form of headache or neuralgia was generally present, together with more or less marked symptoms of mental instability.

On the Continent, hysteria has also been the disease most frequently treated by hypnotism, and successful cases of every known form have been reported. These are so numerous that it is impossible here to give even a *précis* of them. (See also "Hysteria, Treatment of," p. 132.)

IV. *Mental troubles of a hysterical nature. Perversion of sentiment, obsessions, irresistible impulses, hallucinations, melancholia, etc.*

Perversions of sentiment are of common occurrence in hysterical patients, and are generally associated with insomnia, depression, uncontrollable weeping, etc. The changes in sentiment vary from indifference or dislike for those formerly loved, up to a more or less intense hatred, sometimes associated with impulses to injure the person disliked. Once hypnosis is induced, such cases generally quickly recover.

Hypnotic treatment often gives good results in cases of obsession. The following are some amongst the varieties which have come under my notice :—Involuntary contraction of the bladder, arising solely from a fixed idea as to urination, and independently of distension and other physical causes; dread of cancer, followed by delusion as to its presence; fear of insanity, associated with insomnia and melancholia; excessive blushing, with fixed idea of being constantly noticed and adversely criticised; fixed idea of being haunted, with delusions as to visits from the spirit of a deceased enemy; fixed idea as to the possibilities of having injured others accidentally, with constant *folie du doute* and *délire du toucher*, together with suicidal impulses and more than one attempt at suicide; fixed ideas as to business incompetence, with resultant withdrawal from society; morbid fears as to death, accident, loss of employment, associated with agoraphobia, delusions, hallucinations, and suicidal impulses; dread of thunderstorms; claustrophobia; dread of travelling by rail or carriage; fixed idea that something dreadful would happen to the Almighty if anything were done by the patient contrary to various forms of popular superstition; fear of catching syphilis, with *folie du doute* and *délire du toucher* associated with constant washing of the hands and cleaning, or destruction, of personal effects; impulse to touch certain objects, with dread of some fearful catastrophe if this were omitted; fixed idea of sexual incapacity, associated with consequent impotence. All the cases just referred to recovered, while some of them have passed ten years without relapse.

The mental condition in the above cases varied widely. In many instances the patients fully recognised that the fears or ideas which tormented them were absurd and groundless. In others the morbid ideas were more or less completely assimilated, the fears had become delusions, and were often associated with hallucinations.

Successful cases of obsession are also reported by Schrenck-Notzing, Hecker, Wetterstrand, van Eeden, de Jong, van Renterghem, Bernheim, Delbœuf, Gorodichze, Russell Sturgis, Voisin, Burot, Mavrokakis, Bourdon, etc.

VICIOUS AND DEGENERATE CHILDREN.—At the International Congress of Hypnotism, Paris, 1889, the therapeutic value of suggestion in the following diseases of children was generally recognised: Incontinence of urine and fæces, nervous "tics," nocturnal terrors, chorea, onanism, blepharospasm, and many other disturbances of the nervous system of a functional character. Further, that suggestion was an excellent auxiliary in the education of vicious and degenerate children, especially where there were habits of lying, cruelty, theft, inveterate idleness, cowardice, or imbecility.

I have seen several children—who undoubtedly suffered from moral insanity—recover under hypnotic treatment, and some of them (after the lapse of many years) show no symptoms of the original disease. Some were dirty, insolent, violent in temper, and addicted to self-abuse and purposeless theft, and, in addition, suffered from nocturnal terrors, insomnia, and various "tics."

According to Bérillon, hypnotism is not only useful in moral perversity, but also forms the best method of treating various nervous tricks. He draws special attention to the frequency of nail-biting in children, and to the fact that it is often associated with other marks of degeneracy. Such conditions can be successfully treated by suggestion; and I have seen instances of nail-biting, even when the habit has been long established, that have disappeared after a single hypnotic *séance*.

Liébeault records 77 cases of enuresis nocturnalis, 45 being boys and 32 girls. The youngest was three, the oldest eighteen years of age, the average being about seven. In all, with the exception of 9, the malady dated from birth. Of these, 56 recovered, 9 were improved, while in 12 there was no alteration. In my own practice, the results, though not so striking as those of Liébeault, have been eminently satisfactory.

NEURASTHENIA.—Schrenck-Notzing has published a more or less detailed account of 228 cases of neurasthenia treated by hypnotism, and the following, amongst others, report successful cases, Brügelmann, Bérillon, Bourdon, Voisin, Burckhardt, Forel, Ringier, Ritzmann, Bourru, Burot, Stadelmann, von Corval, Michael, Drozdoivski, von Kozuchowski, Neilson, Bernheim, van Renterghem, Wetterstrand.

According to Schrenck-Notzing, the sexual perversions so often associated with neurasthenia, even when these have changed the entire personality, are frequently cured by suggestion.

INSANITY.—According to the late Dr. Auguste Voisin, of the Salpêtrière, no one believed it was possible to hypnotise the insane until 1880, when he first succeeded in a case of acute mania. From that date, until his death, Voisin devoted himself largely to the treatment of mental disorders by hypnotic suggestion; and, while he admitted that hypnotism could do nothing in somatic mental affections, such as general paralysis, cerebral softening, etc., stated that he had cured many cases of undoubted insanity, as well as other disorders more or less closely allied to it, *e.g.* melancholia with hallucinations, suicidal impulses, and delusions of persecution; acute and subacute mania; hysteria and epilepsy; morphinomania; dipsomania; obsessions, such as *délire du toucher*, agoraphobia, claustrophobia, etc. Voisin regarded hypnotic suggestion as extremely valuable in mental disorders, and often obtained good results with comparative facility once hypnosis had been induced, a fact which caused him to express his regret that all the insane were not hypnotisable.

Dr. Répoud (Cantonnal Asylum of Marsens, Fribourg) claims to have cured cases of undoubted insanity, while successful cases are also reported by Burckhardt (Préfargier Asylum), Liébeault, Brémaud (Brest), Velander (Yönköping, Sweden).

In 1890, Drs. Percy Smith and A. T. Myers published an account of the hypnotic treatment of twenty-one insane patients in Bethlem Hospital. Improvement followed in six instances, but the results appear to have been due more to increased personal attention than to hypnotic influence.

Dr. George Robertson, in 1893, hypnotised and controlled the worst case of suicidal and homicidal mania there had been in Morningside for ten years. He concluded that, in insanity and allied disorders, hypnotism was of service in producing sleep, quietening the brain, and preventing outbursts of excitement from passing into mania; also in dispelling fleeting delusional states and minor psychoses, as well as for purposes of management.

In 1897, Dr. Woods, Hoxton House Asylum, reported good results in fifteen cases of insanity; the majority of these, however, apparently belonged to minor forms of melancholia.

Personal Results.—With very few exceptions my results in cases of *undoubted* insanity have been disappointing. In many instances I have failed to induce hypnosis, while in others, although I was able to produce sleep and relieve the pain of organic maladies, the suggestions were entirely without effect upon the insane ideas. There were, however, some favourable exceptions, *e.g.* a case of acute mania with attempted suicide, followed by mutism, insomnia, violence, and persistent refusal of food. The patient, who had been certified, was in an asylum, and all feeding was forcible. After the first *séance* forced feeding was never necessary, and the patient made a complete recovery.

I have also obtained good results in subacute puerperal mania with suicidal impulses, and in cases of melancholia of a more or less hysterical type. In some of these cases the patients had previously been certified.

Voisin's claim to have been the first to employ hypnotism in the treatment of the insane is untenable. In 1852, Esdaile hypnotised thirty-seven insane patients at the Calcutta Asylum, but admitted that the therapeutic results were disappointing. About the same date, Dr. Keen (Berhampore Asylum, India) found hypnotism of much service in procuring quiet and maintaining discipline, thus forestalling the observations of Dr. Robertson. Braid and Elliotson also reported cases of insanity cured by hypnotism.

Dipsomania.—Hypnotism yields excellent results in dipsomania. Amongst my successful cases, of both sexes, the duration of the disease had varied from five to seventeen years; some had had several attacks of delirium tremens and epilepsy, and in most there was a family history of alcoholism. In many instances repeated and prolonged treatment in retreats had failed, while the "gold cure" had been equally unsuccessful. Some of the earlier cases have now remained over ten years without relapse.

Forel claims to have obtained excellent and durable results in dipsomania, and successful cases are also reported by Voisin, Ladame, Tatzel, Hirt, Neilson, de Jong, Liébeault, Bernheim, van Eeden, van Renterghem, Wetterstrand, Schrenck-Notzing, Krafft-Ebing, Hamilton Osgood, Cruise, Tuckey, Woods, etc.

Much depends, however, upon the patient's mental attitude and the operator's management of the case. The following are the more important points:—

1. The patient must be willing to be cured. Some, amongst my earlier failures, afterwards confessed that they had been forced to come by their relatives, but never wished to be cured, and had persistently opposed my suggestions.

2. If possible all stimulants ought to be stopped the moment treatment begins. When a patient is taking a bottle or more of spirits a day, this cannot be done without risk of delirium tremens. In such cases, the quantity taken should be rapidly diminished, and, unless the patient is deeply hypnotised, the insomnia, sickness, etc., which are apt to arise should be combated by drugs.

3. It is important, especially at the commencement of the treatment, that the patient should not be left alone. He should always have some trustworthy person near him, to whom he can confide his temptations, and who will aid him in overcoming them.

4. The operator must be persevering and not easily discouraged; patients, who ultimately do well, sometimes relapse more than once during treatment.

5. Even when the craving disappears quickly, the patient ought to be hypnotised regularly for a month, and afterwards occasionally for a few weeks.

6. As a general rule, the deeper the hypnosis the greater the chance of success. I only know of one instance amongst my own patients where a relapse followed after somnambulism had been obtained. On the other hand, many have recovered when hypnosis had only reached the stage of drowsiness, while in some instances the recovery of the patient was the only evidence of hypnosis.

7. During hypnosis distaste of alcohol ought to be suggested to the patient, as well as the abolition of the craving. Finally, he must be made to understand that he can never look forward to being a moderate drinker, and that the only choice before him lies between total abstinence and drunkenness.

Morphinomania and other Drug Habits.—Morphia and other drug habits frequently yield to hypnotic treatment. In many of my cases of neurasthenia and hysteria, especially where insomnia was a marked symptom, a drug habit had been established. Some took morphia regularly, either by the mouth or hypodermically, while a yet greater proportion used chloral, sulphonal, etc. One patient, who suffered from hysterical neuralgia, frequently inhaled chloroform in addition to injecting morphia. In these cases the disease which had given rise to the drug habit was treated first; if the patients recovered, they often spontaneously gave up the habit or it disappeared in response to further suggestion. Thus, the patient, who had long taken chloroform and morphia, abandoned both as soon as his neuralgia ceased.

A cocaine habit sometimes results from the prescription of cocaine spray for hay asthma or other affections of a like nature. One of my patients, who recovered under hypnotic treatment, had for eight years used an ounce of cocaine per week in this way.

Wetterstrand, amongst other cases of drug habits, cites 38 in which morphia was injected subcutaneously; of these 28 recovered. Many of the cases were extremely grave and of long standing, and with several the abstinence treatment had been tried without success—sometimes more than once. Most of them were treated in private houses; the morphia was never stopped at once, but rapidly decreased, while the patient was constantly informed of the diminution.

Dr. Fulda, Frankfurt-on-Main, reports a case of morphinism of ten years' duration, which was cured after three months' hypnotic treatment; two years later there had been no relapse.

One of Dr. Marot's patients had taken morphia for six years. She could not pass half an hour without giving herself a subcutaneous injection, and her arms, legs, and abdomen were covered with numerous cicatrices and subcutaneous nodules resulting from the use of the syringe. She recovered after hypnotic treatment, and three and a half years later there had been no relapse.

Successful cases are also reported by Voisin, Bérillon, Tanzi, and many others.

EPILEPSY.—Elliotson, Esdaile, and Braid reported cases of epilepsy said to have been cured by mesmeric or hypnotic treatment. In one, cited by Elliotson, there had been no relapse after two years, and in one of Braid's after eight years. Both patients had apparently suffered from genuine long-standing attacks of *grand mal.*

Wetterstrand asserts that some of his earlier patients have now remained many years without relapse, and his general results are so startling that Forel said he only accepted them after he had personally investigated Wetterstrand's work.

More or less successful cases are reported by de Jong, Bérillon, Stadelmann, Woods, and others.

With the exception of a case of epilepsy, associated with dipsomania, in which five years have passed without relapse, my results, although encouraging, are not conclusive. Out of eleven cases marked improvement occurred in five instances, but complete recovery in none. In one case of four years' duration, in which there were several attacks of *grand mal* per week, and much impairment of intellect, there was a remission for twelve months; the patient then relapsed, but the fits ceased under renewed treatment. In another somewhat similar case the attacks ceased for eighteen months, then a solitary one appeared, while now twelve months have passed without a further attack.

The cases already cited by no means exhaust the therapeutic applications of hypnotism, and the following are some—amongst the many widely differing diseases—in which it has been employed with success:—

MENSTRUAL DISORDERS.—According to Braid, hypnotic treatment was the best for regulating menstruation, and for curing or relieving morbid conditions connected with it. Later writers hold similar views. Thus, Forel cites cases in which he personally succeeded in causing the appearance or cessation of the period during hypnosis, and also in regulating its duration and intensity, while, in other instances, he produced the same results by post-hypnotic suggestion.

The following cases are recorded by Tyko Brünnberg, of Upsala:—

	Cases.	No Improvement.	Improved.	Cured.
Amenorrhœa . . .	9	1	2	6
Menorrhagia . . .	9	3	2	4
Menorrhagia and Dysmenorrhœa . .	5	1	1	3
Dysmenorrhœa . . .	3	1	1	1
Total . . .	26	6	6	14

One of the patients, a girl over 20 years of age, had never menstruated, but did so after the second hypnosis.

Some of the successful cases of dysmenorrhœa were severe and of long standing, the periods being much lengthened and the intervals shortened.

Grossmann records a case of amenorrhœa, of three years' duration, in a woman 30 years of age. A few hours after the first hypnosis, the period commenced slightly, and became copious after the second *séance* next day.

The following case is recorded by Bernheim:—

Madame M., aged 35.—Up to the birth of her first child, menstruation was regular, with an interval of 21 days; afterwards, she suffered from menorrhagia and dysmenorrhœa, the interval between the periods varying from 11 to 13 days. Bernheim suggested a progressive retardation of the periods, a definite time being fixed for the appearance of the menses, *e.g.* 25, 27, 28, and 29 days. These suggestions were successful; an interval of 29 days was established, the dysmenorrhœa ceased, and the duration of the period was reduced from 6 to 3 days.

Successful cases of amenorrhœa, dysmenorrhœa, and menorrhagia are also cited by Delius, Wetterstrand, Voisin, Bérillon, Gascard, Journée, Marandon de Monthyel, Bourru, Burot, Dècle. Many of the cases were of long standing, and had resisted other methods of treatment, while their recovery was confirmed by later reports.

My personal observations accord with the conclusions just cited. In nearly every instance, where deep hypnosis had been induced, the dysmenorrhœa ceased, even when it was associated with various forms of uterine displacement. In many cases of menorrhagia, the length of the interval was increased, and that of the period diminished. Some of these patients, who formerly had to pass at least a week in bed at each period, have now no interruption in the activity of their lives.

In one case of amenorrhœa of eighteen months' duration, the period appeared a few hours after the first *séance*, and again a month later, when hypnosis was reinduced. In another, of eighteen months' duration, the period appeared at the day indicated, in response to a post-hypnotic suggestion made three weeks previously.

In the majority of cases, the hypnotic treatment required was short, and the menstrual functions remained normal after its cessation.

Secretion of Milk.—Several cases are recorded in which the secretion of milk has been increased or arrested by suggestion. One of the earliest is cited by Esdaile. His sister-in-law, when weaning, suffered from an accumulation of milk in her breasts, which rendered them painful and swollen. Esdaile hypnotised her; in half an hour she was free from pain, while next morning the breasts were soft and comfortable, and without further secretion of milk.

An experiment, the reverse of this, was related by Braid. He hypnotised a patient and suggested an increase of milk in one breast. On waking, she had no recollection of what he had said, but complained of tightness in the breast. Her child was fourteen months old, and her milk had almost disappeared; despite this, the breast became almost immediately distended with it. A few days later, Braid successfully repeated the experiment with the other breast. The patient suckled her child for six months longer, the supply of milk being more abundant than it had been at any time since her confinement.

The following case is reported by Grossmann:—B., aged 20, primipara, suckled her child for a fortnight; then ceased to do so as she had to leave home. Three weeks later, she returned and wished to nurse the child again, but she had no milk in the right breast and hardly any in the left. The patient was hypnotised, and the sensations associated with the flow of milk suggested. In three minutes, the veins of the left breast became enormously congested, and milk began to flow from it. At first, repeated pressure failed to produce a single drop from the right breast, but when the suggestions were repeated it was secreted freely.

An interesting case is recorded by Freud, where a patient was unable to suckle her child, not from want of milk, but because violent vomiting followed whenever the child was put to the breast. This symptom had appeared after a previous confinement, and the patient had been compelled to abandon her attempts to suckle. On the present occasion, and also on a subsequent one, hypnotic suggestion rapidly removed all difficulty.

STAMMERING.—As a rule, good results depend upon deep hypnosis, but exceptions occur, and cure may take place when slight hypnosis alone has been induced. Thus, one of my patients, an inveterate stammerer, whose infirmity rendered him self-conscious, shy, and miserable, completely recovered although he barely reached the stage of lethargy. All the other morbid symptoms disappeared when the stammering ceased, and the patient has recently been called to the bar. Wetterstrand reports 48 cases, average age fifteen years, with the following results: recovered 15, improved 19, no improvement 14.

SEA-SICKNESS.—Slight hypnosis has apparently little effect in preventing sea-sickness. The results, however, are often very successful when profound hypnosis can be induced. Here, even when the suggestions have been made months previously, sickness is often entirely prevented, even with patients who had previously been invariably ill on short and calm voyages. One of my patients, who before treament had been a phenomenally bad sailor, afterwards made several voyages to and from India, without sickness or rehypnotisation.

RHEUMATISM.—Grossmann records 123 cases of chronic articular rheumatism, and claims to have obtained good results in every instance. Tatzel, Delius, and others, also report successful cases. As far as my personal experience goes, the pain of all forms of rheumatic affections, both acute and chronic, can be relieved or abolished by suggestion in deep hypnosis. In one instance, a commencing attack of rheumatic fever was apparently aborted by keeping the patient in the hypnotic trance for forty-eight hours. In two cases of rheumatoid arthritis good results were obtained, even although slight hypnosis alone was induced, *e.g.* Mr. ——, aged 45. Illness of two years' duration. Joints swollen and distorted; walked badly, use of hands and feet much impaired, and suffered from insomnia and severe pain. Careful treatment at home and at foreign baths had produced no improvement. After hypnotic treatment all morbid symptoms, except those due to organic changes in the joints, disappeared and the patient resumed work.

Miss ——, aged 26.—Illness of seven years' duration. All the joints of the extremities were affected, knees ankylosed, elbows and wrists almost immovable. Patient unable to walk, or even to move in bed; she invariably slept on her back, and could not turn on to her side. The stiffness in the hip joints prevented her sitting; she was in constant pain, and suffered from emaciation and amenorrhœa. Result of hypnotic treatment: Patient now strong and well nourished. She sleeps well, is free from pain, and menstruation is regular. The absence of pain enabled passive movements to be made, and now, although there is still much stiffness and distortion, the patient can move freely in bed, walk with crutches, sit up with comfort, and use her hands for various forms of work.

Deafness.—The range of hearing can be increased in the healthy individual by suggestion; many instances are also reported in which improvement or recovery has taken place in cases where the hearing had been more or less impaired by accident or disease. In the majority of those I have observed, there was a history of scarlet fever with otorrhœa. In most cases the perforation of the drum had healed, and there was little to be observed in the way of physical abnormality. The hearing, however, was defective on one or both sides, the watch being heard sometimes only at a distance of an inch or two, at others only on pressure. When deep hypnosis was induced, the results were frequently striking, and the hearing became normal or even hyperæsthetic. Several years later the improvement was still maintained.

In cases of total deafness—congenital or otherwise—no improvement was obtained.

In Menier's disease the results are interesting. In a typical case, there had been sixteen years' deafness of the right ear and two years of the left. At first, the patient had suffered from occasional attacks of headache, giddiness, etc., these being followed by an increase in deafness. Later, there was marked vertigo, the patient was unable to walk unaided, and this was associated with rotatory movements, constant headaches, and numbness of the hands and feet. In all cases where deep hypnosis was induced, there was a slight improvement in hearing, with partial or complete disappearance of all the nervous symptoms. A year later, these patients reported that the improvement in hearing had not been maintained, but that there had been no return of the nervous symptoms.

Skin Diseases.—The following cases are from my own practice:—

Mrs. ——, aged 49.—Pruritus vulvæ and eczema of four years' duration, unrelieved by treatment. Intense nocturnal irritation, insomnia, and chronic constipation. The uterus was retroflected and bound down by adhesions; this, together with the constipation, was supposed to have originated and maintained the disease. The sphincter ani was stretched under ether by Mayo Robson, but this neither cured the constipation nor relieved the other symptoms. Hypnosis was induced at the sixty-seventh attempt. Result: Rapid and complete recovery, with no relapse after three years.

Miss ——, aged 15.—On the back of the left arm, just above the wrist, a patch of skin, 2½ inches long by 1½ broad, was the seat of constant perspiration, which had existed from early childhood. This was always excessive, saturating the bandages which enveloped the arm, and dripping upon the floor. Hypnosis, with somnambulism, was induced at the first attempt, and next day the perspiration had markedly diminished. The patient was again hypnotised, when the perspiration ceased, and two years later there had been no relapse.

Successful cases are reported by other writers. Thus, Hamilton Osgood cites (1) a case of severe and intractable eczema of nine years' duration in a boy, aged eleven. Hypnotised. Recovered. (2) Mrs. ——, aged 88, severe eczema of nine years' duration. Hypnotised. Recovered. (3) Mr. ——, aged 60, severe and painful eczema of forty years' duration. Hypnotised. Recovered.

Therapeutic Summary.—*Insanity.*—Forel, while admitting the genuineness of Voisin's therapeutic results, asserted that his so-called insane patients simply suffered from hysteria. The brain, he says, is the instrument we employ in suggestion, and if the instrument itself is spoilt, we are no longer able to make use of it, or only to a very small extent, as a means of reacting upon itself and influencing its functions in general.

In reply to this, Voisin, while denying that all his cases were hysterical, claimed that the successful treatment by suggestion of that class alone, marked a therapeutic advance of no little importance. Our asylums, he said, contained a large number of hysterical insane, who were violent and dangerous, and who inspired both disgust and pity by their tendency to drink and steal, and by their lies, dirtiness, obscene and unnatural acts. Many of those, who had long been asylum inmates, were, thanks to hypnotism, now leading active and useful lives.

Later, Forel himself reported a case of delusional insanity, which he had successfully treated by hypnotism. The question whether insanity in its graver forms can be cured by hypnotism is still an open one, but one must not forget, in endeavouring to estimate the value of this form of treatment, that the experiment practically dates from Voisin's first case in 1880, and that comparatively few attempts have been made by others to give the method a fair trial. The value of the Bethlem cases is much lessened by the fact that in no instance did the number of attempts to induce hypnosis exceed nine, while in six cases only one was made.

A certain proportion of the insane are cured or recover, and it seems reasonable to suppose that a method of treatment which, without drugs, can produce sleep, remove excitement and other morbid mental conditions, may prove a valuable remedial agent.

Epilepsy.—Here, the results are encouraging, but not conclusive. In some instances, hystero-epilepsy may have been mistaken for genuine epilepsy, while in others, sufficient time has not elapsed to enable us to judge of the permanency of the cure. On the other hand, if other medical men have not obtained successes equal to those claimed by Wetterstrand, few, if any of them, have employed his special method, viz. prolonged hypnotic sleep.

Other Diseases.—Apart from insanity and epilepsy, the therapeutic results must be regarded as satisfactory, and the following are some of the more important points as to the statistics on which these results are based:—

1. The cases observed have been numerous. Thus, to take a few examples, van Eeden and van Renterghem treated 1089 persons between the years 1887 and 1893, while Wetterstrand's cases up to 1893 amounted to 6500. Liébeault has published no complete record of his thirty years' practice, but, from 1887 to 1890, he treated 1756 cases. From 1882 to 1886, Bernheim attempted to hypnotise 5000 of his hospital patients, while a few years later the number had risen to 10,000. In the first year of his hypnotic practice, Forel treated 205 patients, many of

whom were insane. In 1898, he informed me that he had tried to hypnotise about 1000 persons, and had succeeded with over 95 per cent.

2. Many of the observers, notably Forel, had already gained distinction not only in medicine, but in other departments of science.

3. The majority of the observations were checked by others. Thus, Bernheim's cases were watched by Beaunis and Liégeois, while the majority of Voisin's were hospital patients, who were under the observation of keenly critical and sometimes hostile colleagues. Further, the cliniques, even when these were not hospital ones, were always open to other medical men, and I have spent many weeks amongst the patients of Liébeault, Forel, Wetterstrand, van Eeden, van Renterghem, Bernheim, etc.

In the therapeutic section of this article successful cases are alone cited. Naturally, however, hypnotism, like other methods, has its failures. These are due to many causes, amongst which the following may be cited:—(1) The selection of unsuitable cases. Organic diseases, which do not respond to other forms of treatment, are equally incurable by hypnotism. In such maladies, however, some of the symptoms are due to functional disturbances, excited beyond the actual limits of organic lesion, and these are often relieved by hypnotism. (2) Hypnotism is rarely resorted to until all other forms of treatment have failed. The disease is thus engrained, as it were, and the patients have the fixed idea that they cannot be cured. Recovery, under such circumstances, gives a special significance to hypnotic statistics. (3) The time allowed is often far too short. Patients who for twenty years have tried other methods in vain, grudge giving a few months to hypnotism, while others regard the treatment as a sort of operation, which they condemn as unsuccessful if it does not cure at the first sitting.

In judging hypnotic results, therefore, there are three things which ought specially to be remembered. (*a*) The cases treated in this country are almost invariably those which have been unrelieved by other methods. (*b*) Even under these unfavourable circumstances, the percentage of success is high, and many functional nervous disorders, which apparently receive little or no benefit from drugs, are relieved or cured by hypnotism. (*c*) Further, with due care hypnotic treatment is absolutely devoid of danger. The will is strengthened instead of weakened, and the patients soon learn to rely upon themselves instead of upon the operator.

Hypnotism in Surgery

Esdaile justly claimed that painless mesmeric surgery was common in his hospitals, years before the days of ether and chloroform.

The following cases from my own practice illustrate hypnotism as an anæsthetic:—Miss A., aged 20.—Double strabismus. Operated on by Bendelack Hewetson, 1889. Hypnotic anæsthesia alone. The patient turned her eyes as directed, so as to put the muscular fibres on the stretch. Later, she sustained a severe fracture of the nose. Hypnosis was induced and the bones moulded into position. There was no pain during or after these operations.

Mr. B., aged 40, 1890.—Fracture of bones of arm and shoulder girdle, with injury to soft parts and subsequent ankylosis of joints. The adhesions were frequently broken down under chloroform. Result, swelling, inflammation, and renewed immobility. Ultimately, the patient refused the anæsthetic, and would not allow passive movements without it. He was hypnotised at the first attempt, and the adhesions broken down without pain; this was repeated from time to time, and followed by complete recovery.

The late Mr. A. Turner, L.D.S., published an account in the *Dental Journal* for March 1890, of some of his operations on my patients. He stated that on one occasion he had extracted forty teeth, selecting those cases likely to afford a severe test. Hypnosis was induced and terminated almost instantaneously; anæsthesia was complete, and yet the movements of the patient could be controlled by the operator, thus obviating the necessity for gags, etc. A weak, anæmic girl, with valvular disease,—likely to "deepen" under nitrous oxide, or to collapse for a day after ether,—was quickly and quietly rendered unconscious. Two left and two right molars, and a lower bicuspid, all difficult teeth, were painlessly extracted and without subsequent suffering.

On 28th March 1890, a number of my patients were operated on at Leeds, and, according to Mr. Pridgin Teale, who was present with sixty other medical men, "the experiments were deeply interesting, and had been marvellously successful."

The following were two of the cases:—

C., aged 19.—Dental caries. A weak, anæmic girl. Hypnotic anæsthesia was produced by my written suggestion, and sixteen teeth extracted, while I remained in another room.

D., a boy aged 8.—Exostosis of great toe. Hypnosis first induced two days previously. Mr. Mayo Robson removed the great toe nail, then the exostosis, and part of the first phalanx.

In none of the cases was there any pain either during or after operation. Some were nervous subjects, highly sensitive to pain in the normal condition; others were strong, healthy working men. All returned to Goole the same day, and in every case the healing process was remarkably quick.

Further operations are reported by other observers, thus:—Tillaux, colporrhaphy; Schmeltz, ectropion and amputation of breast; Bourdon, uterine fibroid; Grossmann, fractures and dislocations; Sandberg, dental operations; Forel, cataract; Wood, necrosis of humerus; van Eeden and van Renterghem, dental operations.

Granting, as recent statistics apparently show, that 94 per cent of mankind can be hypnotised, it must still be admitted that many preliminary attempts are required in a large proportion of cases. Further, hypnosis frequently never becomes deep enough for operative purposes; this is specially noticeable amongst the nervous and hysterical. Under these circumstances, unless there were grave objections to the use of other anæsthetics, I should consider it a waste of time to attempt to hypnotise a patient for operative purposes alone.

Apart from this, hypnotic anæsthesia possesses many advantages, thus:—(1) Once deep hypnosis, with anæsthesia, has been obtained, it can be immediately reinduced at any time; (2) No repetition of any hypnotic process is necessary, the hypnotiser's written or verbal command is sufficient; (3) The hypnotiser's presence is not essential, the patient can be put in touch with the operator by written order, or by other means previously suggested during hypnosis; (4) No abstinence from food or other preparation is necessary; (5) Nervous apprehension can be removed by suggestion; (6) Hypnosis is pleasant and absolutely devoid of danger; (7) Hypnosis can be maintained indefinitely and terminated immediately at will; (8) The patient can be placed in any position without risk—a not unimportant point in operations on the mouth and throat—and will alter that position at the command of the operator; (9) Gags and other retentive apparatus are unnecessary; (10) Analgesia alone can be suggested, and the patient left sensitive to other impressions—an advantage in operations on the mouth and throat; (11) There is no tendency to sickness during or after operation, a distinct gain in abdominal cases; (12) Pain after operation, and during subsequent dressings, can be entirely prevented by suggestion; (13) The rapidity of the healing process, possibly as a result of the absence of pain, is frequently very marked.

HYPNOTISM IN OBSTETRICS

Elliotson and other mesmerists of his day reported many cases of painless confinement, and in their time newspaper announcements of births were sometimes followed by the words "painlessly during hypnotic trance." Many instances of painless hypnotic birth are reported by modern observers; amongst others, the following cite cases in which all the patients were primiparæ—Kingsbury, Schrenck-Notzing, Wetterstrand, Mesnet, Fraipont, Fanton, Dobrolsky.

Dr. Bourdon suggested to a patient, in labour at the full term, that the pains should cease and not reappear for eight days. He claims that the experiment was successful, and that the confinement took place painlessly at the time he had selected.

Schrenck-Notzing states that hypnotic suggestion can render labour painless. It also facilitates its course by regulating the position of the body and limbs, and increasing or diminishing the uterine contractions through the action of the voluntary muscles.

Dr. Fanton asserts that suggestion acts directly upon the uterus, and that the operator can cause its contractions to appear or disappear at will. Thus, uterine inertia may be successfully combated without ergot, and the use of forceps frequently rendered unnecessary. The after-contraction of the uterus can be excited and maintained by suggestion, and post-partum hæmorrhage prevented; while, in cases of adherent placenta, turning, etc., the uterus can be relaxed and the necessary operations facilitated. Dr. Fanton claims to have excited premature labour by suggestion, and considers it criminal to have recourse to other means—dilatation of the cervix, perforation of the membranes, etc.—without having first attempted this method.

Although Dr. Fanton founds his statements on numerous cases and experiments, many of them observed and confirmed by other medical men, the results he claims are so startling that few are likely to accept them without further evidence. One must admit, however, that the involuntary contractions of the uterus are sometimes arrested by emotional states. Thus, when a patient is in active labour, her pains may suddenly cease and remain in abeyance for hours, if a stranger comes to attend her instead of her own medical man.

Pregnant women are more easily hypnotised than those suffering from nervous diseases; and from this, and other reasons, there are fewer objections to the employment of hypnotism in obstetrics than in general surgery. Notwithstanding, unless the methods of inducing hypnosis are greatly improved, its use as an anæsthetic will ever remain restricted. Hypnotic anæsthesia, however, must always be of keen interest to both physiologist and psychologist. It is a thing apart, not an ordinary narcotic, illustrating familiar methods of preventing pain by checking all conscious cerebration. It is a new departure—the first successful attempt at dissociating forms of sensation, which throughout the known history of the human organism have almost invariably existed together. Here, pain is suppressed by the inhibition, from among all the subject's possible sensations, of precisely those which would in any way be disagreeable to him, while general consciousness may be unimpaired, and sensations, other than painful ones, rendered more acute by suggestion.

STAGES

The hypnotic condition is divided by different observers into a varying number of stages. Most of these classifications, particularly Bernheim's, are complicated and misleading, while none are quite satisfactory, as the stages themselves are more or less the result of training, and thus artificial. The following classification is the one I follow:—

1. *Slight Hypnosis.*—Changes in the voluntary muscles can be induced. Memory unaltered.
2. *Deep Hypnosis.*—Here, in addition, changes in the special senses can be evoked, and there is sometimes partial amnesia on waking.
3. *Somnambulism.*—In this stage, all the phenomena of the two previous ones can be evoked, and sometimes also others (appreciation of time, etc.), which cannot be produced in the earlier stages. The chief characteristic of somnambulism, however, is the amnesia which follows it, *i.e.* the waking consciousness retains no recollection of what has occurred in hypnosis.

The division of the hypnotic state into different stages is of little practical importance to the physician. With rare exceptions—such as the production of somnambulism for operative purposes—none but curative suggestions are given, and, in the majority of the successful cases, recovery takes place before any deep stage is reached. With a certain proportion of such patients, the phenomena of deep hypnosis might be evoked and classified, if suggestions were directed to that end. Experimental and therapeutic work, however, should be kept absolutely distinct.

SUSCEPTIBILITY

In 1892, Schrenck-Notzing published his *First International Statistics of Susceptibility*, which showed that fifteen observers in different countries had tried to hypnotise 8705 persons, with the following results:—6 per cent were uninfluenced; 15 per cent became somnambules, while 78 per cent were less deeply hypnotised. These figures are fairly representative, although certain observers have obtained still better results. Thus, out of 1756 consecutive cases, Liébeault's failures only amounted to 3 per cent, while Wetterstrand only failed in 3·7 per cent out of 3209 cases.

Susceptibility is little affected by race, and hardly at all by sex; but children are more easily influenced than adults. According to Forel, every mentally healthy person is naturally hypnotisable, while the majority of observers agree that the hysterical and ill-balanced are the most difficult to influence.

The causes which affect susceptibility adversely are too numerous to be referred to in detail; the most important are fear and disquieting emotions of a like nature. Thus, the psychical preparation of a patient for hypnosis is as important and complicated as the physical preparation essential to antiseptic surgery.

In the first 500 cases, drawn from my general practice, there were only two failures, while 48 per cent became somnambules. This susceptibility, far above the average, was apparently due to the following causes:—I had already gained the patients' confidence by attending them in previous illnesses. At that time hypnotism was not discussed in the public press, and the patients had not been frightened with absurd stories as to its dangers. Many of the patients were healthy persons hypnotised for operative purposes, while the percentage of cases of long-standing nervous disease was small.

In the first 100 cases, drawn from other sources, the failures reached 22 per cent. Here, most of the patients suffered from serious and long-standing nervous disease. All were strangers to me, while the majority had heard that hypnotism was dangerous and inevitably associated with loss of consciousness and volition.

REMARKS ON METHODS AND THE MANAGEMENT OF THE HYPNOTIC STATE

Some further hints, as to the induction of hypnosis and the management of the hypnotic state, may be useful to those about to practise hypnotism.

1. Do not attempt to hypnotise your patient at the first interview. Hear his history; form your diagnosis, and make friends with him. Then tell him that what you propose doing involves neither interference with volition nor possibility of harm. Explain that he will have to rest quietly, with his mind fixed on some monotonous idea, while you make suggestions, and that these, when given under specific circumstances, tend to arouse powers which are latent in his own brain, and which may be used for his benefit. Above all, emphasise the fact that only a very small proportion of those who are cured pass into a condition in any way resembling sleep.

2. At the next visit, place the patient in a comfortable chair and darken the room. Then, after making him gaze fixedly upwards for a few seconds, tell him to close his eyes, and to keep them voluntarily shut until you request him to open them. Meanwhile, verbally suggest that he shall pass into a more restful and drowsy state each time he visits you, and lose all his morbid symptoms. The latter suggestions must be varied according to the nature of the case; you must also explain to the patient that they are not likely to be realised at once, and that frequent repetitions are often necessary.

Sometimes patients are easily hypnotised at the first attempt, and respond at once to curative suggestions. With the majority, however, the length of treatment required bears some proportion to the duration of the disease and the mental instability of the patient, and I have sometimes only succeeded in inducing hypnosis after a hundred failures.

Curative suggestions, repeated in a more or less varied form, are usually given for ten minutes to half an hour at a time. According to Wetterstrand, however, too much importance has been attached to suggestion, while the health-giving action of hypnotic sleep has not been sufficiently recognised. He records many cases of hysteria, epilepsy, morphinomania, etc., where the patients were kept in the hypnotic state for a month or more. Nurses, put *en rapport* with them, fed them at stated intervals, while the action of the bladder and bowels was regulated by suggestion. The results were extremely satisfactory, and no untoward symptoms of any kind occurred.

This method is only applicable to cases of deep hypnosis and somnambulism; fortunately, however, the majority of subjects respond to curative suggestions as soon as the slightest hypnosis has been induced, and thus Wetterstrand's method of prolonged sleep is rarely necessary. In some instances, however, it succeeds when ordinary hypnotic methods have failed. Thus, one of my patients, who had suffered from insomnia, spinal neuralgia, dysmenorrhœa, and menorrhagia, was cured of the three former conditions by suggestion. The fourth was unrelieved, and the patient had to remain in bed a full week out of each month. She was kept in the hypnotic trance for three days before a period, and then for five during it, while frequent suggestions were given. Since then the interval, duration of the period, and amount of loss, have become normal, and the patient, instead of remaining in bed, can now take active exercise without inconvenience.

HYPNOTIC THEORIES

First, to summarise the phenomena requiring explanation: The somnambule recalls the events of normal as well as of hypnotic life, but, when hypnosis is terminated, remembers those of ordinary life alone. Further, he can influence and control his own mind and body to an extent undreamt of by the waking will. Finally, there is no impairment of consciousness or volition, nor, in proper hands, the slightest danger.

In slight hypnosis memory is unchanged, but an increased power of controlling the organism always exists.

(*a*) *The Mesmeric Theory.*—According to the mesmerists, the phenomena were due to a vital fluid or physical force, which was transmitted from operator to subject. This force also existed in certain metals, crystals, and magnets, but all were not influenced by it; those who could be were termed "sensitives."

(*b*) *Braid's Theories.*—The subjective nature of the phenomena—the fact that they were due to changes in the patient's own brain—was the essential part of all Braid's theories. On other points, his views changed with increased knowledge. At one time, he attributed everything to an involuntary monoideism; this view was published by John Hughes Bennett in 1851. Here, the phenomena were explained physiologically by the arrested action of some of the "fibres of association." Psychologically by dominant ideas; these had obtained undue influence, because other ideas, which ought to have controlled them, did not arise, as the portion of the brain, with which the latter were associated, had its action temporarily suspended, *i.e.* the connection between the ganglion cells was broken, owing to the interrupted action of the fibres of association.

Later, Braid believed that volition was unchanged in hypnosis, the moral sense increased, and suggested crime impossible. He observed that several phenomena could be evoked simultaneously, and without the subject having passed through any condition resembling sleep. Verbal suggestion excited, but did not explain, the phenomena; the healthy were the easiest to influence, and the condition, as a whole, could only be accounted for by the intelligent action of a secondary consciousness.

(*c*) *The Salpêtrière Theory.*—This, in all essential points, reproduces that of the mesmerists. The old fallacies as to metals, magnets, drugs in sealed tubes, etc., are revived; the subjects, however, instead of being termed "sensitives," are called "hysterical."

(*d*) *Theories of the Nancy School.*—Bernheim reproduces the mental side of Bennett's theory, and ascribes the phenomena to a psychological inhibition. In addition to adopting Braid's discarded view, Bernheim reproduces some mesmeric errors, and adds others of his own. He believes in *rapport*, the possibility of suggesting crime, and the practical identity of hypnosis with normal waking and sleeping life. There is, he says, no special state deserving the name of hypnosis, *suggestion* is everything.

In some recent theories, claiming originality, the physiological half of Bennett's masquerades in modern terminology. Thus, instead of "association fibres" we have "neurons" and "neuronic groups"; "dendrons" contract and break the contact between nerve cells, while a supposed decrease in cerebral activity is explained by an "inhibition of the amœboid movements in the pseudopodic protoplasmic prolongations of the neuro-spongium."

(*e*) *The Subliminal or Secondary Consciousness Theory.*—This theory presupposes an intelligent secondary consciousness, and explains the phenomena of hypnotism by the arousing of powers over which we normally have little or no control, instead of by the arrested action of some of the brain centres which subserve normal life. As William James points out, the principle on which it depends is largely admitted by science, and it gives, what no other theory does, a convenient working hypothesis. The characteristic phenomena of hypnosis show increased, not diminished, mental power. Cerebral inhibitions, no matter how involuntary or incomplete, do not explain improvements in memory, arithmetic, and appreciation of time, and the subliminal consciousness theory marks an important advance towards solving these problems.

LITERATURE.—Hypnotism possesses a rich literature. MAX DESSOIR, in his *Bibliographie des Modernen Hypnotismus*, published in 1888, cited nearly 800 works by 500 authors, and in the *Erster Nachtrag zur Bibliographie des Modernen Hypnotismus*, which appeared in 1890, 382 additional works are recorded. MOLL also has contributed much that is valuable to the same department, and to these two authors readers are referred for detailed biographical information. Several journals, notably the *Revue de l'Hypnotisme* and the *Zeitschrift für Hypnotismus*, occupy themselves exclusively with hypnotism. Others, such as the *Annales de Psychiatrie* and the *Proceedings of the Society for Psychical Research*, contain important contributions, mainly psychological. MOLL's *Hypnotism* gives the best general account of the subject from the scientific side, while BERNHEIM's *Suggestive Therapeutics* is one of the most valuable of the practical works yet published.

Hypoglossal Nerve, The.

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THE hypoglossal or twelfth cranial nerve is the homologue of the anterior root of a spinal nerve. It is interesting to note that in some animals the twelfth nerve possesses a posterior root furnished with a ganglion like a spinal nerve. A similar condition has been met with in a few instances in man (Quain).

The nerve arises from a nucleus which is the direct continuation in the medulla of the multipolar cell columns of the anterior horns of the spinal cord. This nucleus, which lies close to the middle line, in its lower part is situated anterior and external to the central canal. Higher up it comes to lie on the floor of the fourth ventricle, internal to the nuclei of the glossopharyngeal, vagus and spinal accessory (see vol. ii. p. 23). The nerve fibres leave the nucleus in successive bundles, which run obliquely forwards through the substance of the medulla. They lie external to the fillet and pyramid, and eventually appear in the groove between the pyramid and olive, on the anterior surface of the medulla. Ten to twelve of these bundles unite to form the hypoglossal trunk which passes through the anterior condyloid foramen.

After leaving the cranium the nerve descends almost vertically to the lower border of the digastric muscle, lying between the internal carotid artery and the jugular vein. It is then directed forwards above the hyoid bone, and passes beneath the digastric, the lower end of the stylo-hyoid and the mylo-hyoid muscles, while it rests upon the hyoglossus. Finally, by piercing the genio-glossus muscle it reaches the tongue, to which its terminal branches are distributed.

The hypoglossal, which is purely a motor nerve, has, according to Quain, the following distribution: "The hypoglossal nerve proper supplies only the muscles of the tongue, with the exception of the palato-glossus and the pharyngeo-glossus. Fibres derived from the first three cervical nerves which are associated with the hypoglossal for a part of their course, are distributed to the infrahyoid muscles and the genio-hyoid, others of uncertain origin pass to the skull and dura mater and to the internal jugular vein. The hypoglossal forms connections with the pneumogastric, lingual, upper three cervical nerves, and sympathetic."

The hypoglossal nerve is the motor nerve of the tongue, of the depressors of the hyoid bone, and of some of the elevators. Since the nerve is in great part distributed to the tongue, it is in that organ that the more important effects of lesions of the nerve are to be looked for.

When one hypoglossal nerve is paralysed the tongue, when protruded, deviates to the paralysed side. If there is complete bilateral paralysis, the tongue lies on the floor of the mouth and protrusion is impossible. Articulation, mastication, and to some extent deglutition, are interfered with when the tongue is completely paralysed. On the contrary, if the lesion be unilateral there may be little or no impairment of these functions. If a lesion involves the nerve or its nucleus, the tongue wastes and the mucous membrane forms characteristic furrows on its surface, while corresponding diminution occurs in its electrical excitability. Under these circumstances fibrillary tremors are frequently to be seen in the wasted muscle. When the supranuclear portion of the nerve is the seat of the lesion these changes do not occur.

The upper hypoglossal neurons, the nucleus, or the nerve in any part of its course, may be damaged by disease. Outside the cranium the nerve may be implicated in a new growth. Occasionally it is divided in an attempt to commit suicide. A neuritis of the hypoglossal nerve is of very rare occurrence, possibly because the nerve lies deeply and is not exposed. A few cases of supposed rheumatic neuritis have been recorded. In its course within the skull the nerve may be involved by meningitis, tubercular and syphilitic growths, disease of the bone, or neoplasms. Paralysis of one-half of the tongue alone rarely results from a lesion in this situation; other nerves are almost always involved. Within the medulla the nuclei or nerve fibres may be damaged by a tumour, hæmorrhage, local inflammation or softening, when the resulting paralysis is usually bilateral. Degenerative lesions affecting the hypoglossal together with adjacent motor nuclei occur in bulbar palsy.

The comparatively frequent association of hæmiatrophy of the tongue with paralysis of the palate and vocal cord on the same side in central lesions suggests a common origin for the motor supply of these structures. Weakness of the orbicularis oris is often associated with paralysis of the tongue. In hemiplegia unilateral paralysis of the tongue is common; in pseudo-bulbar paralysis, a bilateral palsy more or less complete, and due to a supranuclear lesion, may be met with.

The diagnosis of the situation of the lesion can often be arrived at by a consideration of the associated paralyses.

The treatment must be directed towards the primary disease.

Hypochondriasis. *See* OXALURIA, URIC ACID, INSANITY.

Hypopyon. *See* CORNEA.

Hysteria.—This is described in the following sections:—

1. **General Article on Hysteria.**
2. **Hysteria in Childhood.**
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General Article on Hysteria

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DEFINITION.—Hysteria, as its etymology indicates (ὑστέρα, uterus), was long considered an illness confined to the female sex; the ancients, indeed, regarded it as the result of a disturbance of the matrix seeking relief. Modern writers, however, have with one accord discredited the uterine theory of the malady. The term hysteria, nevertheless, though denied its original signification, is not on that account to be discarded, for, as Janet says, it has such a singularly interesting history that it would be difficult to replace it.

What is hysteria? A precise definition can scarcely be made, as it is not a disease in the ordinary sense, but rather a state of reaction and hypersensibility. If its manifestations are varied, it is no less true that they are all specially allied to affections of the nervous system. Briefly, hysteria is a neurosis of many and perplexing varieties, and of psychic origin, whose disorders often simulate organic disease. Many of the symptoms are variable and merely accidental, others are invariable and form the "stigmata of hysteria." For the sake of lucidity we shall group the symptoms according to the systems or organs of which they indicate functional derangement.

ETIOLOGY.—The appearance of a symptom of hysteria generally proves that the malady has already existed for some time, though latent. The name of a *provocative agent of hysteria* is given to any circumstance which suddenly reveals the malady, but the true cause of the disorder is an hereditary predisposition. If the real cause is unique the provocative agents are numberless. George Guinon, in his thesis, devotes several chapters to them. The *moral emotions*, fright, grief, anger, and other psychic disturbances, are the most frequent causes of occasional hysterical affections; and in every walk of life hysterical subjects are equally liable to attacks. *Traumatism* plays a very important role in its development; it acts in the way of a real suggestion, as much by the nervous shock as by purely physical causes, creating a special nervous condition. To hysterical traumatism must be attributed the railway spine, the railway brain, and the traumatic neurasthenia of German authors. All accidents are apt to provoke manifestations of hysteria, as for instance bites, blows, earthquakes, surgical operations, etc. The severity of the traumatism is not so important as the mental impression produced. It is undoubted that *poisons*, both acute and chronic, are powerful agents; the commonest are lead and alcohol, sulphuret of carbon, tobacco, and morphia; these produce ordinary hysteria without peculiar characteristics. *Infection* also gives rise to hysteria, acting, according to Potain, like an internal traumatism. The commonest forms of such infections are typhoid fever, pneumonia, rheumatism, ague, blennorrhagia, and syphilis. Other general maladies, such as diabetes, sciatica, and gout, have the same provocative action. Certain morbid conditions favour the neurotic tendency, such as anæmia, and sexual excess and nervous disorders, such as disseminated sclerosis, tabes, and myopathy.

The true cause of hysteria is manifestly hereditary predisposition. Briquet's statistics prove that in nine cases out of ten hysterical parents have hysterical children. The cases may be traced to indirect as well as direct heredity; the subjects have had epileptic and neuropathic ancestors. A neurosis often passes over one generation. Grasset proves that scrofulitic, consumptive, arthritic, alcoholic, and syphilitic ancestors produce an hysterical race. Hysteria may appear at any age. With men and women alike it is most frequent between the ages of 19 and 25 years, frequently at the crisis of mental and physical puberty. It is rare before the age of 6 or 8 years, and exceptional after 40 years. The researches of Marie, Souques, and Chauffard prove that hysteria is commoner among men than among women in the lower ranks of society, probably because the artisan class is more exposed to traumatisms and poisons (alcoholic, etc.). Women suffer more in the upper and middle ranks. No race or climate is exempt; hysteria seems to have a marked predilection for the Jewish race.

Symptoms.—It will be convenient to discuss these in the following order: (*a*) convulsive attacks; (*b*) motor and sensory disorders; (*c*) visceral manifestations. It is well to bear in mind that in some cases numerous symptoms coexist, while in others the symptoms present themselves in an isolated manner—"mono-symptomatic hysteria."

Hysterical Convulsions

Convulsive phenomena are frequent in hysteria, more especially in women. According to Pitres 82 per cent of cases in women have convulsions, and only 22 per cent in men. In men, however, the violence of the attack makes up for its rarity.

An attack of hysteria may appear in various degrees. A violent attack is called "hysteria major" or "attack de la Salpêtrière," a less violent attack "hysteria minor." The attack itself may present anomalies.

A. *Great Attack.*—Its different phases are shown in the following table borrowed from Charcot and modified by Grasset:—

Premonitory period	Prodromata Aura hysterica	Psychic trouble and hallucinations Disorders of the organic functions Disorders of mobility Sensory disorders
First period, or Epileptoid period	Phase at commencement Tonic phase Clonic phase Phase of muscular resolution	With movement With tetanic immobility
Second period, or Period of contortions and violent movements Clownism	Phase of contortions or disordered attitudes	
Third period	Period of emotional attitudes or plastic poses	
Fourth period	Period of prolongation. Period of delirium. (Delirium, hallucinations, zoopsia, disorders of movement.)	

Premonitory Period.—The symptoms consist of psychic phenomena, followed by somatic phenomena. Several days before the attack the patient is dull and depressed, and desires to be alone. In other instances there is increased nervous irritability, the patient laughs and cries, and is unreasonably agitated. He sleeps badly, and has dreams and nightmares; he has hallucinations attended with anæsthesia, he sees black and red animals, and his hearing is abnormally acute. His will power at this stage is still strong enough to avert or arrest the attack. Other complications coexist with these psychic phenomena, the appetite is irregular and capricious, nausea and vomiting supervene, with hiccough and abdominal distension (tympanitis). The saliva is abundant, the quantity of urine increased, and the patient is troubled with flatulence, laryngic spasms, etc. Palpitation, hurried breathing, alternations of flushing and pallor, sensations of heat and cold, also ensue. Other phenomena are cramps, shiverings, slight trembling of the legs, general weakness, and temporary contractures.

After this prodromatic period the true aura makes its appearance, and marks the commencement of the actual attack. The ovarian aura is the most frequent, and merits a detailed description. The belly becomes painful at the level of the zone occupied by the ovaries; the pain, at first local, spreads over the whole surface of the abdomen, which is now tender to the touch; the disorder then rises to the throat, and the well-known sensation of the globus hystericus is produced, accompanied by palpitation and a sense of tightness which forces the sufferer to tear off his clothing. The "hysteric fit" produces a sensation of suffocation and strangulation. The aura in certain cases begins in the epigastrium, instead of the ovarian zone, and follows the same course. A cephalic aura is marked by beating veins in the temples, singing in the ears, and specks floating before the eyes; such an attack often commences in the back portion of the brain (*clou hystérique*). In men the auræ belong to the testicular zones and the pseudo-ovarian zones.

First Period, or Epileptic Period.—This develops in three phases. In the first phase the aura is followed by shocks; without warning the patient loses consciousness, the breathing stops, the head rolls and falls back, the mouth is open, the face distorted, the eyes convulsed, the limbs twisted, the body heaving up and down. The patient now becomes rigid, the throat swells, the veins are swollen and projecting, the face is cyanosed and puffy, the features are rigid and a little froth escapes the lips. The arms are thrown up, the elbows out, the wrists bent, and the fists clenched; the lower limbs are extended, the knees pressed one against the other, and the feet pointed. The body is usually in a state of opisthotonos, more rarely in a state of emprosthotonos, or sideways in pleurothotonos. This phase of tonic contracture is usually brief, lasting only one or two minutes. Two phenomena are characteristic of it, namely, loss of consciousness and spasms in breathing. This tonic phase is succeeded by the clonic phase, as in epilepsy. The breathing recommences, irregularly and in hurried jerks; the whole body is shaken by rapid and extensive convulsive motions usually more pronounced on one side, the head oscillates, and the belly is distended. After a time the convulsions cease, and a phase of muscular resolution begins; this is the third period of the epileptic period. The patient is stretched on his back, his face contracted and the head sunk, the eyelids rising or falling from time to time, and the breathing regular and stertorous. The patient is now in a state of coma, interrupted by slight shocks and a degree of contracture; this phase lasts from two to five minutes.

Second Period, or Period of Convulsions.—Convulsions appear in two forms: (1) Contortions or clownish attitudes. (2) Emotional attitudes. The phase of contortions is characterised by disordered movements, attempts at wrestling, or trials of strength (clownism). The attitude called the *arc de cercle* appears during this phase, the body raising itself from the bed, and only resting on the head and the heels. The *arc de cercle* is generally anterior, more rarely posterior or lateral; the face is convulsed and grimacing. The movements of the body are more pronounced than in the preceding period, they simulate bowing, etc., and the limbs are distorted. The patient cries and shouts, tries to bite, and becomes so violent that he has to be held down by force. His attitudes correspond to his various emotions. There exists a transition from this phase to the following one, *the phase of emotional attitudes.* The patient expresses by attitudes his mental condition: anger, prayer, etc. Reminiscences of former hallucinations or scenes which occurred before his seizure, such as violin-playing, disputes, etc., powerfully affect his attitudes. When the attack reaches its height a kind of somnambulistic trance occurs, lasting from five to fifteen minutes. As the patient recovers he can often explain his attitudes, for he often remembers the hallucinations.

Period of Delirium.—In this last phase the patient suffers from delirium and hallucinations, greatly aggravated by outside influences, to which he was formerly indifferent. During this later phase of paroxysmal disturbance, hallucinations (zoopsia), paralysis, partial contractures, and epileptic shocks may supervene. Its duration is very uncertain, lasting hours and frequently days. The end of the attack brings with it a feeling of relief, and the patient resumes his normal condition. Accidents are rare, though cases of death have been known, occasioned by respiratory spasms. If the attacks are frequently repeated the condition is more serious, as the case then becomes one of true chronic hysteria (*état de mal hystérie*).

Such is an attack of *hysteria major* as described by the school of La Salpêtrière. Some writers deny its existence, and argue that it is merely a type of the disease artificially engendered by the surroundings of hospital life. This opinion is not tenable; the reality of this form of hysteria is only too true, and a consideration of its symptoms furnishes a key to the other more or less developed forms of hysteria which are constantly met with.

B. *Hysteria minor.*—This is the most frequent form of hysteria, and it also has three periods. During the pre-convulsive phase the prodromatic phenomena differ very slightly from those of hysteria major; the ovarian aura, however, is wanting. The initial spasm seizes the hollow of the epigastrium; and the attack may often be arrested at this crisis. The period of convulsions, after a comparatively short tonic phase, during which one observes the swelling of the neck, the contortion of the face, and suspension of breathing, is followed by more rapid movements, hurried breathing, clenched jaws, and anguished cries. This period of agitation soon yields to one of emotional attitudes, the attacks generally resembling one another. The crisis terminates in the phases of resolution and delirium, during which the patient neither speaks nor moves, and in which fits of crying, singing, or laughing may alternate. After some little repose the patient can resume his ordinary occupations.

Varieties of Attacks.—In many cases the attack is not always typical; it is often modified either by the predominance of one period which obliterates the others, or by the accession of foreign elements. To illustrate, the aura may supervene without loss of consciousness, and may be marked by a crisis of palpitation or of suffocation, or sometimes by hydrophobic phenomena. The epileptoid attack sometimes simulates epilepsy, more especially as to frequency of attacks; or again it resembles Jacksonian epilepsy. Certain subjects suffer from vertigo like epileptics, and in some cases the vertigo simulates Manière's vertigo.

The phenomena may be limited to those characteristic of the second period, namely, to the *convulsive* stage, and it is to this form of attack that maniacal excesses belong. In the third period the emotional attitudes always present the form of ecstatic emotion. In the fourth period the terminal delirium may present itself under various guises, to which the name of *hysterical follies* has been given. These last appear in somnambulistic form, and are always identical in the same patient. Lastly, the subjects may feel themselves impelled to exhibit the phenomena of *ambulatory automatism.* We must also refer to the *fits of sleep* which simulate coma, but are distinguished by the rigidity of the limbs and by trismus; they are attended by difficulty in breathing, which may be quickened or suspended, or assume the rhythm of Cheyne-Stokes. During this sleep the patient hears sounds, but is incapable of response. This hysterical sleep may be compared to attacks of pseudo-syncope and hysterical fainting fits, and to the irresistible tendency to sleep which is so difficult to distinguish from narcolepsia. In several cases the attack resembles an apoplectic fit (*apoplexie hystérie*), and is accompanied by a hemiplegia either fleeting or permanent.

SENSORY DISORDERS

Sensory disorders constitute one of the invariable and most conclusive symptoms in the diagnosis of the neurosis; they affect equally the mucous membrane, the skin, and special senses; sometimes by the diminution or abolition of sensibility, sometimes by hyperexcitability or hyperæsthesia.

A. *Anæsthesia.*—Anæsthesia has been long known. Witch-hunters and magistrates in the Middle Ages searched with care for "diabolic stigmata" in persons accused of sorcery, but it is only in modern times that the true significance of stigmata has been proved. The anæsthesia shows itself in 60 per cent of cases, but though a common symptom it is variable, and is sometimes complete, sometimes incomplete. Pitres has shown by the following table its different degrees:—

1. Total, bearing on all the sensory perceptions.	(*a*) Complete anæsthesia, properly so called. (*b*) Incomplete hypoæsthesia.
2. Partial, bearing only on certain sensory perceptions, the others not being affected.	(*a*) Loss of the sensations of pain, with the preservation of the tactile sensations: *Analgesia.* (*b*) Loss of the sensations of movement, preserving the tactile and painful sensations: *Themoanæsthesia.* (*c*) Loss of the tactile and painful sensations, preserving those of movement: *Anæsthesia with Thermoanæsthesia.* (*d*) The isolated loss of electric sensations: *Electroanæsthesia.* (*e*) The preserving alone of the electric sensations: *Anæsthesia with Electroanæsthesia.*

From a topographical point of view anæsthesia assumes different forms. In exceptional cases it may spread over the whole surface of the body;

generally it is partial and confined to one side of the body, and is then hemianæsthesia, which has a marked predilection for the left side, being three times more frequent on the left than on the right side; sometimes it appears in islets, insular anæsthesia; and lastly, it may be segmentary. It may be superimposed on paralysis or contracture, affecting a limb or portions of a limb. Like other hysterical phenomena anæsthesia is variable, and may be spontaneously modified either by some cutaneous irritant, such as electric currents, blistering, etc., or in a convulsive crisis by a suggestion or effort to pay attention. Hysterical anæsthesia in contrast to organic anæsthesia is compatible with the normal exercise of most of the reflex actions. The patellar reflexes, cremasteric and abdominal of Rossbach, are generally preserved, so also is the pupillary reflex action. Pricks practised on the level of the anæsthetised region are not attended with a flow of blood, but an appearance of a red aureola sets in, like nettle-rash, followed by a flow of limpid serum. Hysterical anæsthesia has another distinctive characteristic; it is attended with no conscious pain to the patient. The results obtained by an exhaustive diagnosis frequently differ even in the same subject; hysterical patients comport themselves like malingerers.

Anæsthesia of mucous membranes is frequent, being exhibited mainly in the mouth and pharynx, and to a less extent in the genital and anal mucous surfaces.

Ocular Anæsthesia is the commonest form of sensory anæsthesia. It may result in the sudden total loss of vision in one or both eyes, but this is rare; more frequently it causes no subjective visual disorder, but there is sometimes a central scotoma or even temporary hemiopia. On objective examination there is always found a restriction of the field of vision; this is concentric in character, in some cases symmetrical, in others unequal, one or other side being specially affected. While the restriction of the field of vision is permanent, its limits vary from one moment to another during the attack, due to emotion or fatigue, *e.g.* under prolonged examination. There is no lesion at the back of the eye on ophthalmic examination. Besides limitation of the field of vision there is partial colour blindness, chromatopsia. In pictures the patient can distinguish the perspective, trees, etc., but cannot see the colours; everything seems gray. Dyschromatopsia is very common; the loss of the colour-fields is elective, and is generally in a given order, the colours usually disappearing in the following order: violet, green, blue, yellow, and last of all red. The colour-fields of vision are inverted. Whereas in the normal condition blue has the widest field, in the abnormal condition red is the greatest. More rarely the disappearance of the colour-fields is in a different order, viz. violet, green, red, yellow, blue, and this modification of the colour-fields is a very important symptom in hysteria. Although dyschromatopsia is met with in other maladies, the order of colour disappearance is not the same. Thus in alcohol poisoning the red and green go first, and in tabes the red first. In the diagnosing of dyschromatopsia these facts have considerable value. Monocular dyschromatopsia presents one interesting feature: the patient affected with monocular dyschromatopsia for one colour sees this colour correctly by the use of binocular vision, a fact proved by the researches of Parinaud and Pitres. The limitation of the field of vision is accompanied by marked symptoms of asthenopia; if any object is looked at steadily the eyeballs burn and throb, and are wearied.

Other important symptoms appear side by side with these ocular disorders, *e.g.* diplopia or monocular polypia, megalopsia and micropsia. Monocular polypia is discovered as follows: a pencil is placed vertically near the patient's eye (the other eye being closed), and then is slowly removed ten or twenty centimetres; the patient perceives a second object on the temporal side as the pencil is moved farther off; later the two objects may recede and a third appear. This polypia is largely due to a defect in accommodation. These ocular phenomena always accompany anæsthesia of the conjunctival mucous membrane and more rarely anæsthesia of the cornea.

There is *anæsthesia of taste* when the gustatory sensations are deranged; the sense of taste may be limited, perverted, or abolished. As an example of perversions salt may be mistaken for sugar, etc.

Anosmia is rarer than *ageusia*, and may be absolute or partial, or elective, where certain odours are perceived and others unperceived. The general sensibility of the lingual and nasal mucous membrane is sometimes increased and sometimes diminished. *Disorders of the auditory nerves* are sometimes subjective, sometimes objective. In the former, whistlings and singing in the ear may be present, and in the latter total or partial deafness. Rinne's test gives a positive result, and Schwalach demonstrated that the perception of sound lasts longer than when the patient is in his normal state. There are also variations of hearing; a patient may hear a whisper at some distance off, and he cannot hear a watch close to his ear. This perverted acuteness of hearing may be a very pronounced feature. Disorders of the aural organs, in common with those of the ocular organs, are irregular and variable. They often coincide with anæsthesia of the auditory nerve and tympanic membrane, but this coincidence is by no means absolute.

Anæsthesia attacks not only the superficial surface of skin and mucous membrane, but descends to the muscles, tendons, periosteum, and articular ligaments. The anæsthesia in the latter is evidenced by the fact that an unnatural amount of movement can take place without inconvenience to the patient.

The muscular sense is sometimes intact, sometimes altered; the patient loses the sense of the appreciation of weights, does not feel fatigue or the strain following an effort, and is insensible of the position of his limbs. These muscular troubles betoken a serious alteration in the sensory condition, and indicate some degree of anæsthesia.

B. *Hyperæsthesia.*—This is rarer than anæsthesia, and as a rule is more difficult to recognise; it differs also inasmuch as it causes much suffering to the patient. It may be general or confined to one limb, its area being limited to one or two centimetres square. These zones, called *hysterogenic points or zones,* may be situated in the abdomen, about the ovarian or epigastric region, the groin, the thorax, the breasts, the clavicular region, the spinal column, from the top of the throat to the eyeballs, and the malar region. These zones may be bilateral or unilateral, and they are usually first observed near the anæsthetic areas. An attack of hysteria may be brought on by rubbing or pressing these zones, in which case the zones are called spasmogenic; the same means also may be used to relieve the attack, in which case the zones are called *spasmodic* or spasmofrematic. The same zone can be alternately spasmogenic when it is gently pressed, or spasmofrematic when it is vigorously rubbed. As a general rule the zones situated at the level of the thorax are spasmogenic, and the epigastric and ovarian zones are spasmofrematic. These zones are limited, the bounds of hyperæsthesia are clearly defined, and the zones are liable to appear and disappear spontaneously; they can be momentarily suppressed by distracting the patient's attention, or by suggestion, or the influence of electrisation, particularly of static electricity. At the commencement of the attack these hysterogenic zones are painful, and there is a feeling of constriction and bruising. They may be superficial or deep-seated, and are cutaneous in the sense that gentle friction will excite an attack. These hyperæsthetic areas are also sometimes found over the following glandular organs, the mammary gland, the testicles, and the ovaries; the ovarian zone is the most frequent. The seat of the hyperæsthesia is generally the gland itself, for certain authors have traced out the displacements of this zone as the ovary ascends during pregnancy. Hyperæsthesia, though less constant than anæsthesia, is an important factor in the induction of the attacks, and it is more often the cause of violent convulsive attacks.

Up to the present we have only taken notice of the objective disorders of hyperæsthesia, so to speak, now let us consider the subjective—the different *algies* or hyperalgesias of Pitres. These include a variety of phenomena, and give rise to many painful symptoms. Heptalgia, enteralgia, pleuralgia, ovaralgia, and testicular irritability may be prominent features; ophthalmic megrim is more uncommon, and can be distinguished from the organic form by the absence of hemiopia. Other neuralgic affections may occur, such as sciatica. No matter where the seat of pain may be, hysterical neuralgia can easily be distinguished from ordinary neuralgia. The fits of pain do not come on spontaneously, but are provoked by accidental excitement, etc., and may reach an extraordinary intensity if the descriptions of the patients can be believed.

Cephalalgic hysteria, real or superficial, may be limited to one region (*clou hystérique*). Certain patients suffer intense pain, which returns periodically in the evening or early morning hours, and is sometimes mistaken for syphilitic cerebritis or tumour on the brain. It is accompanied with giddiness, sickness, sleeplessness, etc., which may simulate meningitis (meningitis, pseudo-hysterical). Spinal hyperæsthesia may be accompanied with contracture, and simulate a *mal de pott hystérique;* if it follows a traumatism this symptom may give the idea of a fracture of the spine. Other hysterical manifestations have been mistaken for arthritis, and have induced mistaken surgical intervention. Charcot maintains that arthritis of the knee is the most frequent. In M. Focas's cases the arthralgia attacked the knee thirty-eight times, the hip eighteen times, the wrist eight times, the shoulder four times, and the instep twice. These arthralgias are always accompanied by contractures at the elbow and knee, where the position assumed is one of flexion; at the shoulder there is adduction, which causes a twisting of the trunk. Hysterical coxalgia is one of the most interesting of these arthralgias, as it may closely simulate true coxalgia, and as in the latter, the patient is unable to walk, and the least movement of the articulations causes pain. The limbs may be in a state of flexion or abduction. It requires a close examination to distinguish true coxalgia from hysterical coxalgia. In the latter, percussion of the great trochanter causes no pain in the knee-joint. Further, the sensitiveness of the skin in the corresponding region is modified; the zone of pain assumes the shape of a triangle, whose apex is situated over the end of the sacrum, and extends to the folds of the groin and buttock. Hysterical coxalgia develops more rapidly than ordinary coxalgia, and an examination under chloroform shows no subluxation, indicating an organic lesion; in cases where amputation has been resorted to, owing to an erroneous diagnosis, the articulations have been found to be quite sound.

C. *Dysæsthesia.*—Having spoken of hyperæsthesia and anæsthesia, a few words may be said of the paræsthesiæ. *Haphalgia* is the name given by Pitres to the sensory condition, in which a feeling of intense pain is created by contact with certain substances (such as copper, brass, etc.), which in a normal state only produce the sensation of touch. *Allochiria* is the name Obersteiner gives to the condition in which the patient feels on one side of the body the impressions received on the other side.

MOTOR DISORDERS

These disorders may be permanent or temporary; permanent disorders must be carefully sought for, as they often frustrate efforts to find them. To illustrate, the hysterical patient may have slight paralysis which scarcely affects him, and is only perceptible by the use of the dynamometer. This muscular inertia induces disorders which the patient is unable to account for satisfactorily. For example, his legs give way under him when walking, just as they do in tabes.

Amyosthenia may be local or general, and affects the muscles more frequently on the right side than on the left, but varies from one moment to another, being influenced by the attack, by emotion, and by the æsthesiogenic agents. There is no pronounced modification of the tendon reflexes; they are generally normal, though sometimes weakened and sometimes exaggerated. The muscular feebleness has one striking peculiarity, it is only apparent in voluntary and not in automatic motion. In certain cases the amyosthenia has the appearance of akinesia algera. Another well-known hysterical phenomenon (Lasegue's symptom) must be studied along with amyosthenia, the patient cannot move unless he looks to see what he is doing; for example, C. Bell tells of a woman who could not hold her baby in her arms unless she looked at it. This phenomenon is accompanied by a tactile and muscular anæsthesia of the weakened limbs; in other instances the patient cannot move at night in the dark. Individuals suffering from total anæsthesia cannot stand upright if their eyes are shut; no movement can be made unless the patient is looking at the anæsthetised limb, otherwise it remains in its former position (partial catalepsy). Pitres puts this fact lucidly before us; a movement is begun with the eyes open, and is arrested if the aid of vision is withdrawn. Other disorders of movement may be noted: *syncinæsia*, if the patient is told to lift *one* arm he lifts both; *allocinesia*, the patient raises the opposite arm from the one he is told to lift; *heterocinesia*, where the movement executed is the contrary one to the movement he is shown.

Paralysis and Contractures.—Paralysis, contractures, spasms, etc. are the temporary motor disorders of hysteria. They affect both the organic and voluntary muscles; disorders of the organic viscera will be studied in connection with the affection of the viscera, and only voluntary motor disorders will be here described.

Paralysis and contractures proceed from the same causes, and occur either at the end or during an attack. Very occasionally they precede the attack, in which case they are transient, but if they accompany the convulsive attack they may be much more obstinate. At times the predisposing cause is some local inflammation (congestion, laryngitis, etc.).

The attacks affect the moral emotions most powerfully. Traumatism is influential in producing paralysis (*hystero-traumatism*); it need not necessarily be very violent, and there often exists very little relation between the intensity of the trauma and the degree of the motor trouble. The development of the paralysis is preceded by an interval of "meditation," during which the sufferer dreams incessantly of the accident of which he has been the victim; at the end of a time, varying from fifteen days to three months, paralysis or contracture appears; the force of the mental shock is thus seen to be greater than the physical, hence the term psychic paralysis, or paralysis of the imagination (*bei einbeildung*).

Hysterical paralysis develops sometimes slowly, sometimes rapidly, sometimes like a blow or ictus. Wherever it appears it has a certain number of ordinary characteristics. (1) It is usually partial, the limbs still retaining a certain power. These paralytic strokes are not always attended by amyotrophies, nor by vaso-motor troubles and œdema. (2) The temperature is slightly lowered about the affected part, the electric reactions are scarcely modified, and the tendon reflexes are normal. (3) Another typical characteristic of this paralysis is the appearance of a segmentary, anæsthetic zone. (4) The muscular sense is numbed. Hysterical paralysis presents many variations, some astonishing in their intensity and duration, and lasting sometimes hours, sometimes years. The termination takes place on recovery, and may be sudden or gradual, or may give place to a contracture. In some cases the paralysis is systematic, and only affects certain movements. Hysterical paralysis is distinguished from cerebral paralysis by its irregularity and the intensity of the sensory disorders; and from organic paralysis by the absence of any considerable muscular atrophy or disorders of the reflexes. The locale of paralysis is varied; with a monoplegia the paralysis is flaccid, and the limb feels inert. The *sensory disorders* are the following: in the upper extremity the anæsthesia is *en gigot*, with a slight encroachment on the thorax before and behind; in the lower extremity the superior limit of the insensibility is marked by a line passing through the fold of the groin, the iliac crest, and the insertion of the gluteus maximus, leaving out the genital organs and the sacrum. Hemiplegia is the ordinary form of hysterical paralysis, which affects most frequently the left side, and is accompanied by the characteristic gait described by Todd: the lower limb is inert and drags like a useless member, and, unlike organic hemiplegia, there is no power of motion or circumduction, and the feet drag on the ground; hemianæsthesia is always observed. The face generally escapes, though occasionally it is affected in the course of hysterical hemiplegia. In some rare cases this reproduces the *Gubler Millard symptom.*

The existence of hysterical facial paralysis was long doubted, but is now an incontestable fact. It may be accompanied by *glosso-labic hemispasm* (*vide* p. 121). It is seldom very acute, being more frequently a paresis rather than true paralysis; it has one outstanding characteristic, viz. a remarkable disconnection in the motor faculties, the patient being able to laugh or cry, but possessing no power of voluntary action. *Hysterical paraplegia* is sometimes observed. It may be associated with a flaccid state of the muscles, or with rigidity, and in the latter case may closely simulate serious organic disease of the spine. It is distinguished from organic paraplegia by the absence of subjective pains and by the particular type of anæsthesia. In certain cases paraplegia simulates tabes, "hysterical pseudo-tabes"; the association of tabes with hysteria is not otherwise an exception, the two maladies often appearing separately on their own account (hystero-tabes).

The occurrence of paralysis of the ocular muscles, although admitted by certain writers, is problematical.

Contractures develop suddenly or gradually like paralysis, and consist in motor weakness and persistent stiffening of the muscles. The contracture involves the antagonistic muscles, and only relaxes under the influence of chloroform. A segmentary anæsthetic zone is present as in paralysis, the reflexes are rarely augmented, and epileptic tumours are rare, though there is hysterical trembling. A common form of contracture is one which is slight and disappears during sleep. Another type is accompanied by considerable hyperæsthesia with spontaneous paroxysms and pain, together with painful anæsthesia and serious disturbance of the general sensibility. Contractures have a tendency to remain; if they improve they may reappear suddenly on the slightest provocation. With the neuro-muscular system in a state of hypersensibility, a very slight moral or physical shock may set up a contracture. In this class of patient the contracture seems to be waiting to be induced; this peculiarity is called *diathesis of contracture.* This contracture easily reveals itself by percussion of the tendons, by massage of the muscles, by faradisation and compression with Esmarch's bandage. *Diathesis of contracture* is not confined to hysteria, but is met with in cases of organic degeneration of the pyramidal tract. Hysterical contractures must not be confounded with simulated contractures; these latter only occur after a great effort, and betray themselves by the acceleration of the respiratory movements. In organic contractures the sensory disorders are slight, the tendon reflexes are always exaggerated, and the fibrillary twitchings never cease. Hysterical contracture even in its external aspect differs materially from one of organic origin; in this latter the rigidity is slight and may be overcome within certain limits, whereas in hysterical contracture the rigidity is such that it cannot be overcome; the attitude of the limb is also more exaggerated, and the flexion more pronounced than in organic contracture. Contractures may assume different types. As in paralysis, hemiplegic and paraplegic, we shall only mark monoplegic and limited contractures; hemiplegic and paraplegic types are always accompanied by a slight degree of paralysis. When the contracture involves an upper limb its aspect is characteristic, the arm is adducted, the elbow bent, the hand may be closed or opened, and the fingers may be placed as if they held a pen. At other times the wrist is strongly bent and the fist closed. In monoplegia of a lower limb the appearance is one of extension; the contracture is often localised to the foot—the hysterical club-foot, in which the toes are sometimes stretched out, sometimes curled in. Contracture of the ankle is rare. Among the contractures confined to a muscular group we must mention the very rare trismus, the glossolabic hemispasm, the contracture of the muscles of the eyes, blepharospasm, and wry-neck, or torticollis.

Glossolabic hemispasm, already referred to, is frequently conjoined with paralysis and gives the sufferer a very singular expression. In repose the mouth is twisted, the nasolabial furrow deepened, the eyebrows lowered, and slight muscular tremors agitate the face from time to time. If the patient is ordered to open his mouth, or to laugh, or put out his tongue, the spasm becomes intense, the nasolabial furrow is curled up, the teeth are uncovered more on the contracted side than on the sound side, the tongue is thrust out with some inclination to the contracted side, or may be pushed against the cheek inside instead of pushed out. The spasm may extend to the muscles of the eyelids and of the neck, and in exceptional cases it is bilateral.

Blepharospasm may exist alone or in conjunction with the glossolabic hemispasm. There are various forms. In the clonic form there is a constant trembling of the half-closed eyelid. In the permanent or tonic form the eyelid is folded and is difficult to close or raise; if the eyeball is examined it is seen to roll from side to side. In the pseudo-paralytic form there is slight ptosis, without wrinkling of the eyelid or brow, and from time to time there are slight convulsive quivers. This false ptosis is distinguished from paralysis of the levator palpebræ superioris by the lowering of the eyebrow on the side where the spasm exists; in true paralysis it is lowered and accompanied with anæsthesia of the conjunctiva and of the eyelid. *Contractures of the eye-muscles,* though rare, do exist; the disorders of accommodation are attributed to contracture of the Brucke muscle, such disorders as monocular polypia, microcephaly, and megálopsy. In convergent and divergent strabismus the contracture of the motor ocular muscles may simulate ophthalmoplegia.

Among other local contractures *torticollis* may be mentioned; in this contracture the twisting of the neck cannot be prevented, a fact which distinguishes it from paralytic torticollis, a much rarer disease. Torticollis, when associated with subjective pains of the mucous membrane, may be mistaken for cervical caries. Contractures of the muscles of the trunk give rise to scoliosis and kyphosis.

The motor affections which we have still to investigate are chorea, tremblings, saltatory spasms, and astasia-abasia.

Hysterical chorea belongs to two classes: one very rare, arrhythmic chorea, in which the gestures and movements are disorderly, as in the chorea of Sydenham; the other more frequent, rhythmic chorea, in which the shocks attack the limbs, the face, the neck, or the half of the body (hémichorea). It may be thus described: a series of regular cadenced shocks, followed by paroxysmal fits, lasting generally from fifteen to twenty minutes, rarely one or several days. These rhythmical co-ordinated spasms reproduce gestures associated with some action, such as dancing (*chorea saltatoria*), swimming (*chorea natatoria*), a smith beating his anvil (*chorea malleatoria*). (See article "Chorea.")

The hysterical tremblings following an attack or fit are generalised or limited, and are characterised by an intense polymorphism, causing them to resemble all the recognised varieties of tremors. Dutil has described several leading types: 1st, A type of rapid oscillations (eight to ten shocks per second), slight but continuous, suggesting alcoholic tremors, and the movements in Basedow's disease (exophthalmic goitre). Sometimes these tremors are associated with other symptoms of exophthalmic goitre, and the two diseases may simulate each other. 2nd, A type with a mean rhythm (5½ to 7½ shocks per second), allied to mercurial quiverings and those of disseminated cerebro-spinal sclerosis; this trembling when it attacks the lower limbs simulates epileptoid tremors. 3rd, Slow tremors with severer shocks (4 to 5½ shocks per second), resembling the trembling of paralysis agitans.

The saltatorius spasm was closely studied by Brissaud, and consists in a series of successive contractures of the extremities with bending of the legs and thighs, following some excitement, slight shock, or agitation. The patient leaps in an odd way when he walks, as if he trod on hot cinders; the muscular force is intact. This spasm is generally bilateral; if the patient is in bed, a slight irritation, percussion of the tendons, or twisting of the foot over the leg, may provoke this spasm. This dancing spasm may be compared to the *paramyoclonus multiplex* and *electric chorea*, as both are in the list of hysterical accidents.

Astasia-abasia may be defined as the suppression or disorganisation of the co-ordinated movements concerned in walking or standing upright. As may be inferred from this definition, it usually affects the lower limbs, and is rare in the upper limbs, where it betrays its existence in difficulties in writing or drawing. The features are very peculiar. The patient can leap, run, or hop, though he cannot walk or stand upright. Abasia may exist alone. There are two forms of astasia-abasia: the one paralytic, in which walking and standing upright is impossible, for the legs give way under the patient; the other ataxic, in which the symptoms only appear when the patient wishes to walk and stand upright. Astasia-abasia has a tendency to return.

Disorders of the Respiratory Organs

Dyspnœa is frequent in hysteria, and manifests itself under the influence of emotion or without any apparent cause. Its aspect is variable, it is sometimes due to a spasm of the glottis, sometimes to difficulty of breathing arising from paralysis or contracture of the diaphragm, more frequently to *tachypnœa* (from 170 to 180 respirations per minute) without uneasiness or acceleration of the pulse. The *hysterical cough* may accompany dyspnœa, or may be an isolated symptom. In not a few instances these symptoms have led to an erroneous diagnosis of tuberculosis. This cough has marked peculiarities; it is rasping, resonant, without expectoration, rarely permanent, and habitually paroxysmal. The fits may be isolated or grouped, and follow always the same rhythm, from 3 to 4 shocks; it is rhythmical, a true equivalent of the hysterical attack, and accompanied by similar phenomena. It is tenacious and fierce, and has special variations of sound, often resembling a dog barking, a wolf howling, a hen cackling, or a dove cooing. These phenomena of laryngitic spasm occur in epidemic form in convents, schools, etc. With these spasms of expiration there may be spasms of inspiration, fits of yawning, sneezing, and uncontrollable laughter.

The disorders of phonation are many and various. In the first rank comes *hysterical aphonia*, which develops suddenly, and is a veritable pure motor aphasia, without mental impairment; it may sometimes be accompanied by deafness and even agraphia. This disorder has a central origin, and is unconnected with any lesion of the muscles of the tongue and lips. It may last a long time, even years. The hysterical stammering which precedes or follows the aphonia results from veritable polysyllabic aphasia, and can, in many instances, be traced to spasms or contractures.

In *aphonia* the patient can only speak in a low voice or whisper, but articulates well. It often happens, when loud speaking is impossible, that the cough is sonorous and singing is possible, and it should be noted these patients can talk in their sleep. There is contracture or paralysis of the vocal cords. *Phonophobia* is less common, and consists in a fear of speaking too loud. In this condition the patient can only whisper, and any louder speech brings on an attack of pain in the larynx.

Disorders of the Digestive Organs

Gastric hysteria is important, and is frequently the forerunner of other visceral manifestations. *Dysphagia* is the most frequent phenomenon, and is due to spasm of the pharynx and œsophagus. Contracture of the œsophagus betrays itself by the food being returned immediately after ingestion. The initial stage of œsophagia is curious, as certain solid foods pass easily, although liquids are rejected. Bouveret describes a strange case, in which the patient had clonic spasms of the muscles of the pharynx repeated 30 or 40 times a minute—a condition which he regarded as hysterical œsophagia.

Hysterical vomitings often result from contracture of the stomach. They begin immediately after the ingestion of food, and are more or less continuous, often painless, and followed by anorexia.

Hysterical gastralgia is caused by an hysterogenic zone in the stomach or gastric region. The reception of food causes violent pain, often in paroxysms, accompanied by palpitation, dyspnœa, retching, and hiccough. This is a real gastric attack, sometimes so violent as to closely simulate a gastric crisis in locomotor ataxy. When there is gastric atony the sensory disorders are slight, food is vomited some time after ingestion, and the patient suffers from a feeling of repletion in the stomach. Hæmatemesis sometimes appears, and when accompanied by pain may be attributed to ulceration of the stomach. This symptom is not uncommon in hysteria, and is called *hysterical hæmorrhagic pituete.* Mathew and Milian have described the rejection from the mouth of a certain quantity of bloody fluid (generally in the morning), red and acid like currant juice, and composed of ten parts saliva to one part blood. The appetite is perverted, sometimes ravenous, more often anorexied. *Anorexia* is the name given to the systematic refusal of food, and in some cases it is a grave and most troublesome symptom. The patient refuses to eat either from caprice or disgust with life, or from other cause which the patient cannot define. Anorexia is usually total, but it may be partial and refer only to certain foods. It is sometimes variable, transitory, and slight, and at other times just the opposite, and is then accompanied by other symptoms, the chief of which is a slight degree of mental excitement. Later, a state of debility follows, the patient keeps his bed in a half comatose state, the muscles of the neck are paralysed, the extremities are flaccid and cold, he becomes emaciated to a skeleton. It has long been matter of belief that hysterical patients can live without eating, but this is erroneous. The state of the digestive secretion of hysterical patients is variable, the gastric juice being sometimes normal, sometimes weakened in its digestive action. (See article "Neurasthenia.")

The intestinal phenomena are, as a rule, less noticeable than the gastric phenomena. Constipation is the rule, but sometimes attacks of paroxysmal diarrhœa coincide with the vomiting. A mucous enteritis may be met with in these patients; eructations and flatulence may be perpetual, and almost rhythmical. Tympanites is specially important from its frequent occurrence and its often mistaken diagnosis. When pronounced, the abdomen is swollen, painful on palpation, and percussion gives a tympanic sound, the breathing is laboured, food is vomited, and the patient may present the appearance of tuberculous peritonitis. In the paroxysmal form the aspect is that of acute peritonitis, the visage is drawn, the vomitings are persistent, constipation is obstinate, and there is a good deal of fever; this is *hysterical pseudo-peritonitis.* At other times the hysterical phenomena simulate fæcal vomiting. Tympanitis may be local, and gives rise to small tumours, and is not infrequently the cause of an erroneous diagnosis of ovarian, gastric, intestinal, or hepatic tumours. Tympanitis also gives rise to pseudocyesis, specially in those women who crave for children, or those who dread having them. An examination under chloroform alone can solve the question.

Intestinal hysteria occasionally takes the form of appendicitis, the *spurious appendicitis* of Brissaud; rectal spasms may simulate stricture of the rectum. Before passing from the digestive disorders we must mention false hepatalgic colic of hysterical origin. By some writers jaundice is said to be caused by a similar spasm of the biliary passages.

GENERATIVE ORGANS AND ORGANS OF SECRETION

Disorders of the Urinary System.—As we shall later study the disorders of secretion, we shall now confine our attention to the phenomena connected with the organs of excretion. Spasm of the urethra is a not infrequent symptom of hysteria, and when attended by slight hæmaturia may be erroneously diagnosed as renal colic. The bladder may be the point from which painful phenomena arise (cystalgia). Incontinence of urine is uncommon; retention of urine is much more frequent, caused sometimes by spasms or sometimes by paralytic anæsthesia of the mucous membrane of the bladder. To test what form of retention exists, catheterism must be resorted to. If the bladder empties slowly, the water only coming drop by drop, paralysis is indicated; while in spasm of the neck, when the obstacle is passed there is a rush of urine. This spasm of the neck of the bladder may be mistaken for stricture of the urethra.

Disorders of the Genital Organs.—In men hysteria is rarely to blame for genital disorders, except testicular anæsthesia. Many writers have shown the possibility of an hysterical epididymitis. In the case of women the severe neuralgic pains of the pelvis described by various physicians ought to be ascribed to neurotic ovaritis. Disorders of menstruation, amenorrhœa, dysmenorrhœa, and membranous metrorrhagia are frequent, and hysterical attacks have an influence on these disorders. Vaginismus may exist alone or conjoined with a rectal spasm; sexual excess is not such a prominent feature as is often supposed. Indeed it is not uncommon in cases of anæsthesia of the mucous membrane to find hysterical patients quite indifferent and insensible to sexual relations.

Disorders of Secretion.—Among these disorders may be named *salivation*, *galactorrhœa*, *hyperidrosis*, general and local, and lastly, *polyuria*, which merits a special study. The polyuria which follows a convulsive crisis has long been observed. It has been suggested that permanent polyuria of an hysterical nature can be induced by hypnotism, and proofs of this abound. The quantity of urine passed is considerable, at least ten to nineteen litres, and as much as twenty-nine litres being passed daily; polyphagia and polydipsia are its usual attendants, and give the impression of diabetes. Polyuria is a serious and obstinate symptom, and is usually worst in men. At other times, instead of being exaggerated, the flow of urine is diminished, there is *ischuria*, sometimes simply oliguria, sometimes almost total *anuria;* even when the suspension of the flow of urine is complete no uræmic symptoms appear. Vicarious phenomena, such as vomitings, sweating, and diarrhœa, are not uncommon, the secretions usually containing an appreciable quantity of acid and carbonate of ammonia.

DISORDERS OF CIRCULATION AND TROPHIC DISORDERS

Hysterical patients often complain of different cardiac troubles, palpitations, and *angina pectoris;* these differ from organic attacks of angina, in that they are the result of excitement and fatigue, and last some time. Sometimes pain with cutaneous irritation is the dominant phenomenon (neuralgic form), sometimes vaso-motor phenomena are the most prominent, the face becoming pale, thin, and livid (vaso-motor form). Such an attack may terminate with definite cardiac phenomena, notably increased pulse-rate with tendency to syncope; in other cases it terminates in a burst of tears or laughter. Other vaso-motor symptoms are common, more especially flushing and pallor and extreme sensitiveness to cold.

Dermographism, often met with in other nervous affections, is well known in hysteria, and is easily demonstrated. If we draw a line along the skin with the point of a pencil an artificial nettle-rash speedily develops. This takes the form of a broad band, the centre of which appears pale and œdematous, with a zone of congestion on either side. This may frequently be demonstrated after a convulsive attack. Certain hysterical subjects are in a state of vaso-motor diathesia analogous to diathesia of contracture. At a more advanced stage the neuro-vascular disorders express themselves by local *asphyxia of the extremities, erythromelalgia,* and lastly *hæmorrhagia.* Ecchymoses may develop under the skin, appearing equally on the ocular mucous membrane (tears of blood), nasal (epistaxis), auricular (otorrhagia), vesical (hæmaturia), uterine (metrorrhagia). To this list of vaso-motor disorders bloody sweats fall to be added.

The trophic troubles are no less interesting; among them are *bullous eruptions* of the pustular variety presenting sometimes the appearance of pemphigus, sometimes that of zonæ or herpes. These have been described as hysterical eczema and herpes. Pigmentary stains sometimes develop after some mental emotion or crisis. But the most important cutaneous trophic disorders in hysteria are œdema and gangrene.

Hysterical œdema is always unilateral and limited to one limb; it is very rarely general, simulating *anasarca;* it appears often in limbs already the seat of hysterical contraction. The skin affected by œdema is smooth and shining, the colour deep blue or violet, constituting the so-called blue œdema; the local temperature is lowered, the œdema is hard and does not yield to pressure. Blue œdema lasts a long time, generally several years. *Hysterical gangrenes* may be symmetrical and attack the extremities; they are most frequently in isolated plaques. Trophic disorders also attack the cutaneous appendages; it is noticed that the hair falls out, and the teeth and nails become friable.

The trophic troubles which attack the uterus deserve special attention. The *hysterical womb* consists in a temporary or permanent enlargement of the organ, coming on suddenly and accompanied by considerable hyperæsthesia of the skin. The colour of the skin may be normal, or red, or even violet. In certain cases considerable œdema is present, and gives the feeling of a tumour. In many instances of hysterical swellings of womb or breast the patient is operated on under the impression that there is a malignant growth; the analogy with cancer is made complete when there is a gangrenous plaque on the surface of the solid œdematous area. This form of trophic disorder, like œdema, may last a long time. Trophic disorders not only attack the skin and subcutaneous cellulary tissues, but even involve the muscles; *muscular hysterical atrophy* is more or less considerable, and is not accompanied in the majority of cases with any modifications of idio-muscular excitability. The electric contractility is generally diminished, but the reactions of degeneration do not often appear in this form of muscular atrophy. This atrophy sometimes develops alone, but more frequently follows hysterical hemiplegia or monoplegia. The whole limb may be affected or only a portion, and it requires a longer time to recover than a case of simple hysterical paralysis. The tendons are also subject to trophic troubles, *fibro-tendinous retractions* may continue after the paralysis and contractures have disappeared; they are sometimes accompanied by the thickening of the soft tissues and necessitate surgical intervention.

Disorders of General Nutrition

General nutrition can be modified in hysteria in certain directions. It has long been believed that during the paroxysms of hysteria, nutrition is carried on under other than normal conditions. This is not the case: the character of the secretions proves that in hysteria the nutrition is normal. But there is some alteration after the attacks; an examination of the urine proves that nutritive changes then differ from those in a normal state. The alterations observed are commonly called the *urinary formula of hysteria.* If the urine collected in the twenty-four hours after the attack be examined, there is found a reduction of the average amount of uric acid and phosphates, with inversion of the formula of these latter. The total solid, which normally averages from 40 to 52 grammes, is lowered to 35-38 grammes; the uric is lowered to 13 gr. instead of being 20 gr.; the phosphoric acid is 1·2 gr. instead of 2·2 gr., as in the inter-paroxysmal period. Whilst in normal urine the soluble phosphates and alkalies are as 1 to 3, after the attack they are as 1 to 1. This modification is called inversion of the phosphatic formula; the same urinary formula is found in attacks of partial or ordinary epilepsy, chorea, delirium, stupor, and other mental disorders.

The study of general nutrition leads us to observe *hysterical fever.* Its existence has been disputed, as a fever can be so easily simulated, but recent researches undoubtedly prove its existence. The variations of temperature are identical with the different known forms—remittent, intermittent, continuous, and sometimes polymorphic, changing quickly from intermittent to continuous. The rise of temperature may be considerable, reaching 41°, and even more. From a clinical point of view there are two very distinct forms: one in which hyperthermia exists alone, without other symptoms—the so-called hysterical fever—often accompanied with convulsive phenomena; the other form is accompanied by certain visceral disorders, and simulates other maladies, such as typhoid fever, meningitis, pulmonary tuberculosis, peritonitis, and malaria. This hysterical fever differs from infectious fevers in its irregularities, the absence of the usual serious symptoms, and its resistance to antipyretics; it should be considered as equivalent to an attack. The diagnosis of hysterical fever is a diagnosis by *exclusion*, but it should only be arrived at after careful consideration, as many organic disorders begin with hysterical manifestations.

Disorders of the Mental State

We have already spoken of the delirium which follows an hysterical attack, and we shall now study the mental condition between the paroxysms. This mental condition is one characterised by modifications of memory and will power. Derangements of memory are frequent, and cause the patient to make conflicting and contradictory assertions. Pierre Janet has made an exhaustive study of *amnesia* and its different modifications. (1) It is systematised, not confined to one given period in the life of the individual, but to a group of recollections; for example, it causes an individual to forget a foreign language or proper names. (2) It is localised, and causes the individual after some severe shock or accident to forget events accompanying or preceding the event. (3) It is generalised; the individual has then to be taught everything like a child. (4) It is anterio-retrograde, that is to say, the individual forgets events as soon as they happen. In spite of this amnesia the intellect remains intact, but the power of attention is enfeebled.

The will is often as much disturbed in hysteria as the memory; automatic actions are unconsciously accomplished, but voluntary actions, such as a new piece of work, are impossible. This loss of will power is very marked in hysteria, and explains the facility with which the patient responds to suggestion. The emotions and affections are often disturbed; sometimes the hysterical patient is excited, easily affected, and exceedingly irritable; at other times he is indifferent, sad, melancholy, and depressed; in both cases the emotional reaction is exaggerated and out of proportion to the cause. Hysterical subjects are sometimes accused of being liars, hypocrites, dipsomaniacs, etc., but these faults ought rather to be placed to the account of the mental degeneracy resulting from hysteria than ascribed to the subjects themselves. The question of legal responsibility in hysterical mental troubles is a difficult one. If criminal acts are committed during a paroxystic crisis the personal irresponsibility is incontestable, but if they take place during an interparoxystic period the personal irresponsibility is not quite so clear.

Clinical Forms of Hysteria.—Hysteria may appear in a convulsive form, or in a monosymptomatic form, in which case stigmata should be carefully searched for to prove the diagnosis. The clinical picture varies according to age; the convulsive reactions are less violent in the old—in whom they exist often in a masked state. The case of children is different, and merits a special description.

Infantile Hysteria.[1]—Infantile hysteria has very special manifestations owing to the brain conditions present in infancy. During infancy the brain develops very rapidly, the convulsive reactions are numerous, and at this period there is a continual growth of new and violent sensations; the nervous system is in a state of constant erythism. Education is an all-important factor in the nervous development of the child, and should the parents be of a changeable and unstable character, and the servants coarse and unintelligent, the child's predisposition to nervous disorders will be intensified. Such causes as abnormal physical growth, over-study, helminthiasis, and teething, all tend to develop these nervous disorders in the predisposed subject. The hysteria of early infancy must be distinguished from that of later years and adolescence. With the nursling many disorders attributed to teething are really hysterical. Not a few nervous disorders encountered in infancy where the disturbance exceeds in intensity the degree of reaction normal at that age, and which cannot be explained by an intoxication or infection, must be regarded as hysteria. If during infancy hysteria is rare, it becomes more common as the years go on, and clothes itself in very curious forms. "Who has not seen," says Graucher, "the little ones of four or five years playing with their dolls, and already showing in their shining eyes, their caressing and feline attitudes, their coquetry, and their malice, a neuropathic tendency only requiring opportunity to develop?" From the ages of eight to nineteen years, the disease may develop with most astounding symptoms, but is of shorter duration than in adults; as Charcot says, "Hysteria has no hold on a child." In grave cases infantile hysteria is monosymptomatic. The most frequently observed phenomena are vertigo, cephalalgia, stammering, fits of yawning, digestive disorders of a gastric nature, polyuria, and astasia-abasia. Trophic troubles such as œdema are not uncommon. Ocular disorders, save the narrowing of the field of vision, are seldom met with; hyperæsthesia governs to the exclusion of anæsthesia. Coxalgia, the malady of Pott, and hysterical torticollis, are habitual manifestations of this hypersensibility. In a less advanced degree hysteria expresses itself by cerebral irritation, dreaming, nightmares, and hallucinations; there are also digestive troubles, vasomotor troubles, and special psychic conditions. Hysterical children are precocious, specially with regard to the arts; their affectivities are active, but changeable, the slightest impression produces intense reactions, either joy or grief. Such children have tendencies to lie and dissimulate, their extraordinary statements should seldom be believed, and they often give false witness in courts of justice. Many suffer from mania, are habitually melancholy, and may commit suicide with little or no reason. A commencing hysteria produces the greatest number of cases of simulated organic disease. Hysteria can imitate hooping-cough, enteritis, and meningitis; certain cerebral disorders such as infantile spasmodic hemiplegia, certain medullary affections such as spinal paralysis, nervous disorders such as epilepsy, and organic disorders such as intermittent fever. It is especially in children that neurosis is best simulated. In its mildest form infantile hysteria only appears in psychic phenomena. Children often have pallakinia, tenacious prurigo, and nettle-rash; they sleep badly or lethargically, start and tremble at the slightest noise, and are frightened of the darkness. Their dispositions are flighty, ungoverned, passionate; they are jealous of their brothers and sisters, have ideas unsuited to their age, excessive sympathies and antipathies. They eat badly, are thin, with hollow eyes, and present often attacks of meningism.

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[1] See also "Hysteria in Children," p. 136.

Diagnosis.—The diagnosis of hysteria depends on the presence of stigmata; these should be sought with great care, especially in monosymptomatic hysteria. Should they be wanting, the special circumstances causing the accidents and the antecedents of the patient will be of the greatest value in the diagnosis. The possibility of an organic lesion has to be carefully considered. It is impossible to show here the differential diagnosis of hysteria and all the disorders it may simulate; we shall limit ourselves to indicating in the following table the differences between hysteria and epilepsy, the malady most often confounded with it:—

Epilepsy	*Hysteria*
It shows itself during the first fifteen years of life. Generally there have been infantile convulsions.	Although sometimes existing in childhood, it oftenest appears at the time of puberty, and during the first portion of the adult life.
The fits follow night or morning; the patient falls without any previous warning, in consequence there are scars on the face and skull of an epileptic.	The attack follows the fall, and is preceded by an aura. The patient is aware of the attack, and does not injure himself.

Epilepsy	*Hysteria*
The beginning of the fit is marked by an initial scream.	There is no initial scream. The cries come on at a more advanced stage.
During the fit it is seldom necessary to hold the patient energetically, his movements are not prolonged.	The patient's movements are so violent and prolonged that it is often necessary for several people to hold the patient.
The loss of consciousness is absolute. The patient cannot possibly speak.	The patient utters exclamations, and sometimes pronounces sentences. The consciousness comes back during the phase of violent movements.
Emotional attitudes do not exist.	Emotional attitudes exist, as if the patient were acting in a dream.
The delirium after the convulsions is unreasoning and impulsive.	The delirium is rational, and can be directed by speaking to the patient.
At the crisis of the attack the tongue is bitten, there is involuntary defecation and micturition.	There is no injury to the tongue, and no involuntary defecation or micturition.
The general health is deranged by these fits, and the condition is grave and feverish.	The frequency of the attacks do not affect the general health. The temperature is normal.
After the fit, torpor, sleepiness, curvature.	The attack ended, the patient shows no morbid phenomena.
From a clinical point of view, one finds after the fit a rise in the rate of the fixed residuum of the urea, chlorine, sulphates, and phosphates, without inversion of the formula of the latter.	From a clinical point of view one finds a lowering of the rate of the fixed residuum of the urea, chlorine, sulphates, phosphates, with inversion of the formula of the latter.
No hysterical stigmata, no permanent narrowing of the field of vision.	Besides the attack, there are stigmata and permanent narrowing of the field of vision.
Bromides have a therapeutic action most undeniably.	The action of bromides is nil, or inferior to what they are in epilepsy.

Such are the chief points of the diagnosis between these two maladies. With regard to the diagnosis of hysteria, we must bear in mind that the disease may closely simulate a great number of organic diseases. In doubtful cases, even where stigmata may be present, it is necessary to make a distinction between minor symptoms and those which ought to occupy the first rank, namely, the narrowing of the field of vision and anæsthesia.

TREATMENT

A. *Prophylactic.*—Hysteria is an essentially hereditary malady, and arises from both direct and indirect heredity. Psychic and physical remedies are necessary to counteract the effects of hysteria in an individual so predisposed. Education and physical exercises may do much to correct a tendency to the neurosis. A boy, for example, should be brought up in the family circle, educated by a firm and equable master; all exciting literature forbidden, and physical exercises, such as gymnastics, riding, swimming, and hydrotherapiæ, encouraged, care being taken that the boy does not overdo these things. When he is older the strain of examinations, etc. must be avoided. The girl even more than the boy requires a wise home education; if the mother is highly nervous she should confide her child to the care of a good governess or judicious relatives, for the sight of the mother's nervous outbreaks is bad for the child and favours the neurotic development. At puberty all over-excitement must be avoided. The period of menstruation, with its psychic and physical changes, is fraught with possibilities for young girls. A severe moral shock may make complete psychic and physical repose necessary. All excitement and romances should be forbidden, also excessive dancing, etc.

Among other causes of nervous disorders alcoholic stimulants must be guarded against; subjects should be warned to avoid stimulants, and should not enter any calling that exposes them to the use of intoxicants. With the prophylactic treatment there arises the question, how far marriage and pregnancy are influential in hysteria. It is an old saying that marriage is the cure for this malady, but this is far from being proved. If the marriage is a happy one, and if the young wife is free from care and sorrow, it will certainly have to all appearance a beneficial result. If marriage, on the other hand, is a source of trouble, its influence will be unfortunate. The family ought, then, to be made aware that marriage is not a cure. If hysterical symptoms have not developed before twenty years of age, hysteria is less likely to be serious. The influence of pregnancy is debatable; the moral influences which surround the subject have much more weight than the mere condition itself. Suckling the infant should be forbidden, as it exhausts the mother, and the milk of a healthy wet-nurse is of far more value to the child than that of an excitable mother.

B. *General Treatment.*—Hysteria being essentially a psychic malady ought therefore to have a *psychic treatment.* The first condition of this treatment is for the physician to have entire authority over his patient, that he may know how to speak to him and secure his confidence. He must have sympathy with his patient's sufferings, and be able to assure him of the efficacy of the means used to cure him. It is for this reason that it is often advisable to separate the patient from all outside influences, and place him under strict medical supervision and nursing. In fact it is better that the patient be completely away from relatives and friends. The duration of his absence should vary according to circumstances, and it should be continued for some time after the abatement of the disorder. As the cure progresses the isolation need be less complete, and the patient may gradually resume his ordinary liberty, taking walks, and by degrees returning to his former mode of life. Unfortunately this isolation cannot always be arranged, as even the friends object to it, but grave cases of hysteria can be cured in no other way. Hypnotism is a valuable aid in the treatment of hysteria, but it must not be considered a panacea: numbers of hysterical patients are not hypnotisable. On the other hand, hypnotism itself is not without danger, for repeated seances act as a form of attack. It is not impossible that a subject who has been under the influence of hypnotism may fall back into the same state under the influence of suggestion, such as the looking fixedly at some brilliant object. Hypnotism in itself is not a treatment for hysteria, but is an adjunct of the greatest value, which ought to be reserved for special emergencies (convulsions, delirium, paralysis, spasms, etc.) The means followed to produce the hypnotic sleep are, steadily gazing at a bright object, listening to a monotonous noise (the tick-tick of a watch), gentle rubbing of the eyelids, pressure of a hypnogenic zone, and simple suggestion. It is during this sleep that the suggestion may be exercised at the moment when the patient is plunged in the somnambulistic trance. (See also "Hypnotism.")

The suggestion to the listening patient deserves to occupy a conspicuous place beside the hypnotic suggestion. The patient benefits greatly by the suggestions of his medical attendant, whether addressed directly to himself or overheard when uttered to someone near him. Very often on hearing the doctor emphatically state the opinion that he is much better and nearly cured, the patient believes it, and half the battle is won. This suggestion is rendered more efficacious by the administration of harmless medicines which the patient believes to be beneficial (bread pills, rye powders, etc.) The blue of methylin in pillules, because of the colour which it gives to the urine, is valuable as a means of suggestion of cure. The external treatment of hysteria in general consists of magnets of metals, electrisation, and hydrotherapia, etc. Metallotherapia is based on the belief that the patient has a special sensibility for one or more metals, which, when applied to the surface of the skin, may modify the anæsthesia and the contractures. This therapeutic measure has often been advocated, but the results derived from it are very disappointing.

The electric treatment plays an important and really efficacious part. Static electricity acts like æsthesiogenic and as a tonic; it can be used in the form of electric baths, or if it is better to localise the electricity, it may be used as an electric blast, or brush, or spark. The electric bath is followed by a feeling of comfort and a disappearance of the anæsthesia and the amyosthemia. The appetite improves, and the digestion is stronger. The duration of the seances should be from ten to nineteen minutes.

Electrical faradisation, with the aid of a brush, acts most favourably on isolated anæsthesia, or when placed over paralysed parts or contractures. In cases where one cannot have recourse to static electricity on account of the want of proper apparatus, generalised faradisation may be used instead with good effect.

Galvanic electricity is inferior to faradised electricity in the cure of hysteria. It can, however, be used beneficially in cases of local trophic troubles, muscular atrophy, and œdema. Hydrotherapia is of the greatest value on account of its æsthesiogenic action. The cold douche is advantageous in battling with anæmia and general debility, as it favours the absorption and elimination of the therapeutic factors. The best mode of administering the douche is to have it poured on the body at a temperature of 8 to 12 degrees for the space of 19 to 20 seconds, taking care not to project the jet of water on the head or the hysterogenous zones. Sometimes the cold douche cannot be endured, and in such cases one must have recourse to the Scotch douche, either without transition, that is to say, passing suddenly from a temperature of 35° or 40° to a very low temperature, or if the patient is very sensitive, with gradual transition. In special cases, wrapping in the wet sheet or patting with wet sheet may be beneficial, but these means should, as a rule, merely lead on to the cold douche.

Treatment by mineral waters is not so useful as hydrotherapia; the most highly recommended waters are, Neris, Divonne, Bains, Plombières, Luxeuil, La Malon, Wildbad, Ragatz, etc.

Regimen.—The diet ought to be tonic and abundant, but overloading the stomach has to be avoided. In grave cases certain physicians employ a milk diet, as in the Weir-Mitchell cure of neurasthenia, namely, 100 grammes of milk every two hours, together with complete rest and isolation. Ordinarily one does not need to have recourse to this régime, but it is always best to choose light foods and to eat often, say four meals a day.

Medicinal Treatment.—There is no medicinal treatment necessary in ordinary cases of hysteria, the internal medication being purely symptomatic. To calm the patient alkaline bromides may be given, which are far from being as active as in cases of epilepsy. Injecting morphia should be avoided, as there is a risk of hysterical patients becoming morphomaniacs. The different preparations of valerian (tincture, extract, ammoniated valerian, and preparations of zinc and quinine) have all the great advantage of not being prejudicial. A number of hysterical patients are anæmic, and in their case preparations of iron and arsenic are very valuable. We need do no more than mention the surgical treatment by oophorectomy or hysterectomy. Its success is problematical, and does not compensate for its real inconvenience.

C. *Treatment of the Attacks.*—When the attack is very violent the patient must be guarded from injuring himself. There are two ways of combating the attack itself.

1. In the first rank among the mechanical aids must be placed the compression of the spasmofrenatrix zone, which must be searched for in the ovarian and epigastric regions. In order to touch the zone the hand must be pressed into the iliac fossa, and then having overcome the resistance of the abdominal walls, it will be sufficient to place the two first fingers on the presumed seat of the ovary in order to obtain the desired effect. Pressure of the breasts and nape may also be used with advantage. By gently pressing the eyeballs also the effect of hypnotic sleep may be tried; and electric shocks at intervals may arrest the crisis.

2. As regards medicines, the inhaling of ether and chloroform must be used guardedly. In very bad cases chloroform may be tried. Bromide of ethyl and nitrite of amyl have also been used for the same object.

D. *Treatment of Particular Accidents.*—Anæsthesia of the nerves and senses can be subdued by brush electricity and static electricity. Paralysis, however, presents great resistance to the remedies employed, and it is necessary to have recourse to transfer suggestion and massage. In cases of contracture the medical intervention should be rapid, acting upon the hyperæsthetic zone by the aid of static electricity, and on the anæsthetic zone by electric faradisation. Hypnotism rarely succeeds. Movement of the contracted limb must be avoided. Any application of apparatus may aggravate the disorder, and there is the risk of permanently disabling the limb, and fixing the contracture psychically. In certain cases after the contracture has disappeared retraction is observed; surgical treatment of these retractions should not be used, otherwise there may be a return of the contracture. Among the visceral disorders of hysteria it is important to notice anorexia, as it is one which may endanger the life of the subject. Its proper treatment is complete isolation. The feeding tube ought not to be used, except as a last resource, for the patient may become habituated to it. The patient must be forced to eat even by threats, and there must be no yielding as to the kind or quantity of food to be taken.

Nature of Hysteria

The question of the nature of hysteria is singularly complicated and difficult to treat. Although the subject has attracted the attention of numerous writers, it cannot yet be said that everything is clear with regard to the explanation of its phenomena.

The ancients—Democrates, Plato, and Hippocrates—regarded this neurosis as taking origin in the uterus. Hippocrates defined it as "a 'suffocation' of the uterus which rises to the heart, to the liver, to the hypochondrium, to the loins: when it comes to the liver it produces suffocation, intercepting the respiratory path which is in the belly. . . . These symptoms occur especially in spinsters and in widows who being young and having had children, remain in expectancy. It is best that they become pregnant; as for the maid, she should be advised to marry." In these words the doctrine of the uterine origin of the disease and its treatment by marriage, which has been so strongly adhered to, is formulated for the first time.

Galen, although admitting the possibility of hysteria in the male, associates this malady none the less with disorders of the uterus. He no longer believes, it is true, in the rising of the uterus towards the hypochondrium, but he regards the symptoms of hysteria as dependent on displacements of the uterus (antero-version, retroversion, etc.), and on retention of the menses resulting in a general intoxication of the organism.

Later, during the Middle Ages, the views of Galen were accepted as facts. Cases of hysteria were not uncommon, and the written and illustrated records from this epoch which remain, reproduce pictures in which the features of hysteria are readily recognised. Under the influence of predominant religious ideas, the hysterical subject was regarded as a sorcerer or as one possessed of the devil. The stigmata of hysteria were the "signs of the devil," and the sole therapeutic measure employed was exorcism. Thus, far from making progress, the explanation of the phenomena of hysteria became more and more obscure. In the eighteenth century the uterine theory is reverted to. Hoffmann attributed hysteria to a spasmodic contraction of the uterus; nearer the present time, Lonzer Villemag in 1802, and Landouzy in 1846, regarded hysteria as the consequence of uterine trouble.

In our own time certain writers, if they do not admit the uterine, still maintain the sexual origin of the disease. Certain surgeons, few in number it is true, do not hesitate in severe cases of hysteria to remove the ovaries in the female and the testicles in the male. The results of these more than doubtful operations do not give support to the sexual theory. Cases have been recorded where true symptoms of hysteria have developed in nervous females who have undergone such treatment on account of local disease.

The theory which considers hysteria as a nervous disorder which may develop in the male as well as in the female, is of relatively recent date. It is to Sennert (1572-1637) and to Charles Lefrois that we owe the conception of hysteria as a disease of the nervous system, a conception still very vague and very erroneous, since the last-named author attributed its symptoms to the presence of a collection of fluid within the head. It is none the less a step forward towards the humoral theory of Sydenham, who has given a perfect clinical description of the disease; for he regarded hysteria as a consequence of debility of the nerves, of impoverishment of the fluids, of a disturbance of the *esprits animaux*.

In the nineteenth century the nervous theory of hysteria gained ground through the works of Georget, Brachet, and Briquetait. "Hysteria," says Brachet, "is a neurosis of the central nervous system, which declares itself more or less suddenly in attacks of general clonic convulsions and by the sensation as if a ball were rising in the œsophagus, at the lower part of which it settles, threatening suffocation." As one sees by this definition, the author has only recognised the convulsive form of hysteria. Through Briquetait the question expanded and the nervous theory evolved. He sought to explain not only the transient convulsive phenomena, but also the persistent symptoms; he showed the part which the emotions play in the disease. "To me," he says, "hysteria is a neurosis of the brain, the symptoms of which consist in perturbation of the vital acts which serve to manifest the affections and the passions." This conception is an advance towards the theory by which hysteria is regarded as a cerebral disease, a disorder of the mind. The credit of showing the important part which is played by the "fixed idea" in the production of the various symptoms of the neurosis, more particularly in traumatic hysteria (paralyses, contractures, etc.), is due to Charcot. He demonstrated the possibility of producing hysterical phenomena by suggestion, and emphasised the existence of a mental state *peculiar* to these patients. He also confirmed from the practical side the theory which regards hysteria as a mental disorder, by bringing forward the results obtained by moral treatment in the cure of the disease. His colleagues—Marie, Gilles de la Tourette, Sougues, Guinon, Pierre Janet, etc.—have, by their researches, helped to elaborate and explain certain points. While mentioning the name of Charcot, it would be unjust to forget the names of Möbius and Strumpell, who with him place hysteria among the psychoses, putting it among those diseases which they call the diseases by representation (*durch vorstellungen*).

Looking back at the chief stages through which the doctrine as to the nature of hysteria has passed, we see it regarded at first as a disease peculiar to the female and of sexual origin, then slowly coming to be regarded as a disorder of the nervous system, organic by some, functional by others. At the present time the leading authorities on neurology and mental diseases regard Hysteria as a mental disorder. We shall now briefly review this theory.

If hysteria is a mental affection it is not to be included in the group of the true psychoses. The symptoms of hysteria have a *particular* physiognomy which distinguishes them. The writers who have upheld the mental theory attribute the phenomena of hysteria to :—(1) a dual personality, and (2) a shrinking of the field of consciousness.

The theory of the dual personality has been advocated by Möbius. It is a deduction drawn from *the rôle of the fixed idea* (Charcot), in the development of the symptoms of the disease. This duplication of the personality doubtless exists in somnambulism and in delirium, but it is more difficult to explain by the adoption of this theory certain symptoms, such as the anæsthesia and the hyperæsthesia, in which there exists no conscious *activity* of the individual. It is this part of the theory explaining the production of the unconscious fixed idea on which it is necessary to insist. A somnambulist has two personalities, two separate psychological existences, of one of which he is absolutely ignorant. Charcot has shown that there exists a very remarkable similarity between attacks of hysteria and somnambulism.

If then we admit that during an attack the *fixed ideas* are more powerful than in the waking state, it is easy to conceive that these ideas are able to originate in the individual and to develop during a state of unconsciousness. Abnormal states of consciousness are produced, limited, it is true, in what is really an abnormal personality, and it is the existence of this abnormal personality which enables us to understand the development of the symptoms of hysteria.

This theory of the part played by the *fixed idea* is shared by a number of writers. Gurney and Th. Myers hold that hysteria is an unreasonable auto-suggestion under the control of the normal consciousness. If this theory be applied it explains satisfactorily the symptoms of hysteria, but does not explain the stigmata, and for this reason certain writers look for another explanation of the latter phenomena. They admit, however, that these are the consequence of an enfeeblement of the cerebral function.

Féré regards the subject of hysteria as being in a permanent state of psychical fatigue, which manifests itself by a diminution of sensation, of motor power, and of the will.

Oppenheim considers that a condition of irritable weakness and an abnormal excitability, coupled with nervous weakness, play an important part in hysteria.

Jolly admits of an extreme weakness allowing of the exaggeration of the emotions. P. Zanet goes farther than the preceding authors, and tries to determine the nature of this mental debility ; he regards it as a defect of attention, attention being maintained with difficulty and easily exhausted.

Whether one considers it from the motor or sensory point of view, it is this want of attention which explains *l'aboulic*, the hesitation which prevents the hysterical subject giving himself up to intellectual pursuits, and at the same time taking in and storing up impressions, and from accomplishing many movements. It may be said that his field of consciousness, on account of its limited extent, can only take in one sensation at a time. This diminution of the field of consciousness prevents the individual elaborating and condensing his thoughts, from assimilating them, from making and transforming them into an integral part of his personality.

Pick of Prague arrived at analogous conclusions when he attributed the persistent symptoms of hysteria to diminished power of attention, of perception, and of movement.

As regards the application of this theory to the explanation of the visceral symptoms: it seems that their mental origin cannot be accepted without question, how does it explain the influence of moral treatment, of isolation, and of symptoms such as anorexia and spasms? Are the persistent vaso-motor phenomena to be regarded as the result of an exaggeration of the *emotions?*

Such, broadly speaking, is the psychical theory of hysteria.

We cannot do better than quote the following formulæ, given by Janet, which form a résumé of the mental theory :—

"Hysteria is a mental affection belonging to the large group of diseases due to cerebral weakness and debility. Its physical symptoms are somewhat indefinite, consisting chiefly in a general diminution of nutrition. It is largely characterised by moral symptoms, the chief of which is an impairment of the faculty of psychological synthesis, an abolition, and a contraction of the field of consciousness. This manifests itself in a *peculiar* manner and by a certain number of elementary phenomena. Thus sensations and images are no longer perceived, and appear to be blotted out from the individual's perception, a tendency which results in their persistent and complete separation from the personality in some cases and in the formation of many independent groups. These series of psychological facts alternate the one with the other or co-exist. Finally, this synthetic defect favours the formation of certain dependent ideas, which develop complete in themselves, and unattached from the control of the consciousness of the personality. These ideas show themselves in affections possessing very various and unique physical characters."

Recently Sollier has propounded a new theory for hysteria which differs considerably from the conception of the Salpêtrière school. It is based on the well-known fact that hysterical subjects (*hysteria major*) sleep little, and yet do not suffer from fatigue. He concludes that these patients are immersed by day in a pathological sleep, which renders natural sleep impossible. It is for this reason that if one orders hysterical patients who have grave symptoms to wake up completely after hypnosis, a series of phenomena are seen similar to those accompanying the natural waking state, with regression of the personality. To conclude from this that the subjects of hysteria are somnambulists or rather "vigilambulists," is not a wide step.

The state of vigilambulism is modified, not only when one orders the patients to awake, but also when one orders them no longer to be aware of their anæsthetic zones. The return of sensation, obtained in this manner, is sufficient to arrest all the symptoms comprised in the somnambulistic state. This method, which at the same time is a therapeutic agent, is called by the author the "method of arousing the sensibility." These alterations of sensation are, in the same way, dependent upon a torpid condition of the cerebral centres, which are indeed asleep. It is easy to explain by this conception the different symptoms of the neurosis, since, according as such and such a centre becomes torpid, different symptoms will result, and in cases where numerous symptoms are present several centres may be asleep at the same time. This doctrine admits that the existence of cerebral localisation accounts for the dynamic troubles of hysteria. It deserves on account of its originality to be placed alongside of the generally accepted theories.

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Hysteria in Childhood

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Hysteria is probably much commoner in children than is generally supposed, and it is a matter of no small importance that its frequency should be recognised, for at this age environment, one of the most potent factors in hysteria, is easily modified and controlled; moreover, education in its widest sense can be carefully planned even in its smallest minutiæ, so as to guard against neurotic tendencies at that period when they are most easily checked.

Age.—There is some doubt as to the earliest age at which hysteria may occur. At least three cases have been recorded in which symptoms attributed to hysteria were present at the age of 18 months, and there have been some very doubtful cases at an even earlier age. Hysterical catalepsy has been seen at 2 years (Mills) and at 3 years (Jacobi), and hysterical analgesia at 2¾ years (Barlow). After the age of 4 years hysteria becomes more frequent. The writer has met with hysterical amaurosis with analgesia at 4 years, and has known severe hysterical contracture to occur at 4½ years. But the majority of the cases of hysteria in childhood occur during the five or six years preceding puberty: of 33 consecutive cases under the age of 12 years, which came under the writer's observation, 23 were in children over the age of 8 years.

Sex plays a far less important part in the hysteria of childhood than in that of adults. The predominance of the female sex which is so striking in adult hysteria is much less noticeable in this condition in children. While the proportion of males to females in adult hysteria is variously stated at 1 to 3 (Pitres), 1 to 10 (Bodenstein), 1 to 20 (Briquet), in children it appears to be about 1 to 1·25.

In the series of 33 children mentioned above, 18 were girls, 15 were boys. It would seem, however, that when boys are affected they are liable rather to the milder manifestations of hysteria, than to the severe forms often seen in girls.

Predisposing and Exciting Causes.—There can be little doubt that in many, perhaps in most, cases the tendency to hysteria is congenital—the child is born with a neuropathic tendency; and in this sense the dictum of Pitres, "on nait hysterique, on ne le devient pas," is probably correct.

The influence of heredity is perhaps even more marked in the hysteria of childhood than in that of adults, and it seems likely that the appearance of hysteria at a very early age may be determined in some degree by the intensity of the neurotic inheritance. Not only hysteria itself, but epilepsy, migraine, and other neuroses in the parents predispose to hysteria in the child. Alcoholism in the parents seems to be an important factor.

But it is often difficult to discriminate between the results of heredity and the results of environment. Childhood is pre-eminently an impressionable age, and the child who is in constant association with an hysterical mother, or spends its early years in frequent fear of a drunken parent, may perhaps develop hysteria apart from any influence of heredity. Moreover, such parents are as a rule but poorly qualified to encourage in their children that control of the will, by the intellect rather than by the emotions, which should be acquired during early life, and the lack of which appears to be at the root of some at least of the manifestations of hysteria.

The frequency of rheumatic heredity is also very noticeable. Apart from chorea, the children of rheumatic families seem to be specially prone to minor functional neuroses, and in a considerable number of the cases of hysteria in childhood there is a family history of rheumatism.

A tendency to hysteria is often fostered by the atmosphere of home life. In particular the solitary child who has to pass its early years in the society of adults, and scarcely ever associates with other children, not only becomes self-centred, and acquires those habits of introspection which are so conducive to hysteria, but too often suffers from excessive attention; its trivial ailments and injuries receive an exaggerated sympathy, and its precocity is a constant theme of admiring approval.

The influence of school life as an exciting cause of hysteria is a point of considerable practical importance. It has happened repeatedly that the onset of the hysterical condition in a child has occurred just at the time when school examinations were at hand, and there is abundant evidence that the pressure of school work, especially the keen competition of examinations in the present day, does act deleteriously upon children of nervous temperament. Chorea, somnambulism, fæcal incontinence, and migraine are certainly in some cases closely related to the mental strain of school work, and there can be little doubt that school work, in the same way, may be the exciting cause of hysteria.

But apart from the mental strain, there are some children upon whom school has a harmful effect for an entirely different reason. These are the extremely timid children to whom school is a daily misery, not because they dislike or are unable to do the work, but because they cannot shake off a fear of the teacher, or in some cases a morbid shyness, which makes all association with strangers, even with class-mates, a trial to them.

School punishments also, especially corporal punishments, have been the exciting cause of hysteria and other functional neuroses in childhood; such severe symptoms as hysterical convulsions have been caused in this way.

It is most important in this connection that it should be realised that one of the earliest manifestations of the hysterical temperament in children is sometimes a condition closely resembling moral insanity. The child is volatile and passionate, perhaps rude or even vicious, and may be almost unmanageable; as such it is in continual disgrace, and is apt to be severely punished. In these children injudicious punishment does nothing but harm, and may bring on a troublesome outbreak of hysteria.

Fright is an occasional exciting cause of hysteria; and this is hardly to be wondered at, for the effects of fright on young children are often very profound, and the wilful frightening of children, whether in punishment or in jest, is a cruelty which cannot be too strongly condemned.

Sensationalism is also an exciting cause of hysteria in children. The thrilling ghost story, the still more unwholesome spiritualistic *séance*, which in one case recorded by Charcot produced severe hysterical symptoms in three children in one family—such things are full of danger for the child with hysterical tendencies. And one cannot pass over the influence of sensational religionism—a very different thing, be it observed, from religion; no small number of cases of hysteria in childhood have occurred in schools, especially convent-schools, or other institutions in which the children were exposed to this unwholesome influence.

Traumatism plays quite as prominent a part in the hysteria of childhood as in that of adults. In the best authenticated case of hysteria at the age of 18 months which has been recorded (Gillette), transient hysterical paralysis of both arms was apparently due to a very slight traumatism of one arm.

Depressing influences and possibly toxic conditions are also occasional causes of hysteria in children. In one child under the writer's care the strain of watching by a sister's deathbed was followed by intermittent hysterical spasm of some of the cervical muscles, and in several cases the onset of hysteria followed immediately after influenza.

Some writers have laid much stress upon masturbation as a cause of hysteria in childhood, and it seems likely that the concomitant excitement and subsequent depression may aggravate hysterical tendencies; but it has to be remembered that masturbation and the grosser forms of immorality which are occasionally met with in children are certainly in some cases, perhaps in most, the manifestation of the neuropathic tendency rather than its cause.

Imitation certainly plays a large part in the hysteria of childhood. As far back as the thirteenth century epidemics of hysterical symptoms occurred, affecting children as well as adults, and sometimes only children. Quite recently epidemics of hysterical convulsions and other symptoms have occurred in schools; and there can be little doubt that this imitation-hysteria is not all mere shamming; auto-suggestion, prompted by the example before the child, probably induces a genuine hysterical condition; at the same time malingering is by no means unknown in childhood.

Symptoms.—It is unnecessary to repeat the general symptoms of hysteria here, for they have been described already in the article on Hysteria in Adults, and there is scarcely a symptom of the adult affection which has not been observed also in children. There are, however, some special features of this condition, as it occurs in childhood, which are worthy of notice.

The early years of the boy or girl who in later life develops hysteria, not infrequently show certain indications of the neuropathic taint. When the hysterical symptoms occur it is often easy enough to see these prodromal indications in retrospect; but for the child's sake it is most important that their significance should be recognised earlier. Certainly from the age of 2 or 3 years these early indications may be noticeable, and it may be that even in infancy the occurrence of infantile convulsions may sometimes, as Dr. Coutts has suggested, foreshadow the neurotic tendencies of later life. Such children are often extremely passionate; from caressing affection they will pass suddenly, if thwarted, to a perfect fury of passion. In other respects also they are extremely volatile, and their behaviour, without being insane, is often unreasonable, and they are in consequence extremely difficult children to manage. Some of them also show excessive timidity or a morbid shyness. They are troublesome too in their feeding, full of fads, "can't eat this," and "can't eat that." They are apt to exaggerate their ailments. Often they are solitary children who do not care to associate with other children, and show no healthy interest in the games and amusements of childhood. As has been already mentioned, sexual immorality is sometimes an early manifestation of the neuropathic tendency, and some of these children, before the appearance of definite hysterical symptoms, have been so vicious in their habits, that they have been expelled from schools as unfit to associate with other children.

As regards the hysterical attack, while in adults it often manifests itself as a complicated grouping of symptoms, in a child it tends to be much simpler. In many cases only one or two symptoms are present, and in the writer's experience motor symptoms—and of these the spasmodic rather than the paralytic—have seemed to be much commoner than sensory.

Localised hysterical spasm is one of the commonest manifestations of hysteria in childhood; in one child there is a sudden jerk of the head to one side, in another a transient spastic condition, shifting at intervals of a few hours from limb to limb, in a third a continuous muscular spasm drawing the foot into a position of talipes, and not very rarely the muscles of phonation and of respiration are affected, and a frequent short, dry, sometimes barking cough or some other curious sound is produced.

In children these spasmodic affections are often rhythmical. In two boys aged 10 years, fine tremors of the whole body occurred, and in one case could easily be produced by suggestion; in another case, a girl, similar fine clonic spasm was limited to the lower jaw; in another girl of 11 years, there was rhythmical belching with a curious gurgling sound, as if air were bubbling down the œsophagus; the symptoms ceased directly the child was put in the horizontal position; the sound was so disturbing that the child had been unable to attend school. Hysterical convulsions, however, the hystero-epilepsy and clownism of major hysteria, are much less common in children than in adults; in 4 of the 33 cases mentioned above hysterical convulsions were present, preceded in one girl, aged 11 years, by the globus hystericus. Sometimes, as in adults, these attacks can be brought on or stopped by pressure on certain areas, the so-called hysterogenic zones, but these are very rare in children. The writer met with one case of hysterical dyspnœa in a girl aged 8 years, in whom the attacks could be stopped by pressure on the umbilical region.

Of sensory symptoms hyperæsthesia is probably the most common in childhood; anæsthesia and analgesia are certainly rare. The limitation of the field of vision, to which so much importance has been attached as a symptom of hysteria, is occasionally found in children, but it is so difficult to test a child's vision with the perimeter that such observations are apt to be fallacious. Hysterical blindness and deafness have occurred but rarely in childhood. Complaint of headache is not uncommon in association with hysterical symptoms in children; and in some cases its daily recurrence at a certain hour, or its disappearance when the child's attention is distracted, or its curiously localised character, or the presence of hyperæsthesia of the scalp, suggests that the headache is really hysterical.

A very important group of cases is that in which the hysterical symptoms closely simulate organic disease, the neuromimesis of Paget. Joint symptoms are perhaps the commonest of these in children. The hip-joint in particular is often affected, and it is sometimes very difficult to differentiate hysterical coxalgia from rheumatic or early tubercular disease of the hip. It may indeed be almost impossible to exclude rheumatism; the hip-joint is one of the favourite sites for rheumatism in childhood, and the objective evidence of articular rheumatism in childhood is often extremely slight; moreover, as already mentioned, hysteria is specially liable to occur in the children of rheumatic families; and, lastly, what began as a slight attack of genuine rheumatism may persist as hysterical coxalgia.

Lateral curvature, early spinal caries, and talipes are all simulated by hysteria, but in these, as in the joint cases, the nature of the trouble is often shown by the sudden onset of the symptoms, the diffuse character of the tenderness over the supposed seat of the disease, the variation or disappearance of the symptoms when the child's attention is distracted, the presence of other symptoms of hysteria, and the rapidity of cure by such "moral treatment" as is effectual in hysteria.

A very puzzling condition is that which has been called "pseudomeningitis hysterica." Several instances of this in childhood have been recorded. The following case may serve as a typical instance. A girl aged 9 years had influenza. The temperature was raised to 101° or 102°, but became normal after a few days. From that time she was drowsy, with obstinate constipation, vomited once, and complained of continual headache in the right frontal region; at one time there was photophobia; and there was great difficulty in persuading the child to take food. The child was apathetic, scarcely ever spoke unless questioned, and just before the writer saw her the pulse was reported to have fallen to 45, and the respiration had been of the Cheyne-Stokes variety. The child was thin and extremely weak; she lay curled up on her side, with the face turned away from the light. The heart acted irregularly, the abdomen was much retracted. There was no irritability, the child answered questions readily, there was no stiffness of the neck and no affection of cranial nerves, the discs were normal. Meningitis was suspected, but a few days later analgesia appeared below the knees, and on being removed to hospital and treated with electricity the child was well in about ten days.

Diagnosis.—For the general diagnosis the reader is referred to the previous article on Hysteria. The diagnosis of hysteria in childhood must be approached with the utmost wariness. The fact that no obvious cause can be found for aches or pains or other symptoms is not sufficient reason for labelling them hysteria. Where there is any doubt it is safer as a rule to assume that the condition is not hysterical than to risk the serious error of supposing a case of organic disease to be one of hysteria. Hysteria, moreover, is sometimes associated with or started by an organic lesion; hysterical joint affection, for instance, is sometimes started by a slight articular rheumatism.

The difficulty of diagnosing hysteria in children is further increased in some cases by the fact that only one symptom is present; the so-called "stigmata" of hysteria are commonly absent.

Prognosis.—The immediate prognosis is probably better in children than in adults. It is very rare for hysteria in childhood to resist treatment obstinately as in later years; and as a rule the symptoms disappear in a few days, or at most in a few weeks, under suitable treatment. But the neuropathic tendency remains, and therefore relapses, usually perhaps with some new symptoms, are apt to occur. Moreover, a study of severe cases of hysteria in adult life shows that in many such cases there have been symptoms of hysteria in childhood, and on this account the prognosis as to the future must be guarded, for in some cases at least, hysteria in a child is the indication of a strong neuropathic taint, which is likely to show itself in graver manifestations in later life.

Treatment.—*Prophylactic.*—There are few conditions in childhood which call for more experience and tact in management than the early manifestations of the hysterical tendency; and by this is meant not the fully developed hysteria which is commonly recognised as such, but that less-defined group of prodromal symptoms, noticeable almost from infancy, to which attention has been drawn above. Home life, companions, amusements, education, every detail of the child's life must be carefully supervised, so as to avoid those harmful influences which have been mentioned already as predisposing and exciting causes of hysteria. The choice of a nurse is a matter of the utmost importance. To deal with such children successfully requires more than ordinary tact in the nursery; they need a nurse who is quiet and firm, who knows when to take notice and when not to take notice, when to sympathise and when not, and above all, one who never loses her patience.

In most cases it is probably wisest to send such a child to school as soon as it is old enough, rather than to have a governess or tutor at home; for the mixing with other children helps to counteract the tendency to become self-centred, which is often noticeable in these children, and, moreover, gives them a wider range of interests. But the conditions of school life must be properly regulated; anything like brain-worry—and this is possible at a very early age—must be carefully avoided. The best beginning is undoubtedly the kindergarten, and for a child of 4 years, an hour and a half in the morning may be quite sufficient. The teacher should be informed of the child's peculiarities, and it is essential that a school should be found where the teacher has the necessary tact and patience to avoid scolding and punishment, which are worse than useless for these children.

In these, even more than in other children, education must include not merely mental, but also physical training, whether it be by a well-regulated gymnasium, or by Swedish drill, or by dancing, or by some other form of disciplined exercise.

The amusements and interests of the child must not be neglected, for they undoubtedly exercise a considerable influence on the development of hysteria; like the "idle hands," the idle mind is likely to come to mischief, and it is for those who have the care of these children to encourage in them a healthy interest in games, animal pets, the tending of flowers, simple botany, entomology, or some such occupation.

When the home environment is so unfavourable that judicious management cannot be expected there, it is sometimes advisable to send the child away altogether to more wholesome surroundings.

The general health must be carefully considered, for any depressing cause favours the onset of hysteria; the tendency to vicious habits must be remembered; masturbation in particular, an early indication of the neuropathic taint in girls as well as in boys, must be checked, for it may precipitate the onset of hysterical symptoms.

Therapeutic.—The treatment of hysterical symptoms in childhood is the same in principle as in adult life, and it is unnecessary to repeat it in detail here. Change of environment is one of the most powerful remedial measures. Many children speedily recover when removed to hospital or to some place where they are among strangers and strange surroundings. A change from town life to the varied interests of sea-side or of farm life is often helpful, and such measures may be more effectual than drugs.

Cold douching, massage, and electricity are of value in particular cases.

The influence of suggestion is probably more often of use in children than in adults; an impressive assurance that the symptoms will speedily disappear considerably assists recovery. Moreover, in children, such aids as promises of pleasures conditional on the disappearance of the symptoms are more often available, and are very valuable in treatment.

The use of hypnotic suggestion for children suffering from hysteria has been advocated by Bérillon, but some observers have stated that mental disturbance has followed its use, and on the whole it may be doubted whether it is ever advisable to make use of hypnotism for a condition which, in childhood, is probably always curable by other methods. It may, however, be useful for those children in whom vicious habits, especially masturbation, cannot be checked in any other way.

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HYSTERIA from the surgical point of view, and as I understand it, is that condition in which there is imagination, imitation, or exaggeration of pathological affections. It occurs mostly in females and persons of nervous temperament, and is due to some nervous derangement which may or may not be pathological. Hysterical affections often closely resemble pathological conditions, the explanation of this not being always easy. Sydenham says, as quoted by Holmes, "In whatever part of the body it be seated, it immediately produces such symptoms as are peculiar thereto." Mimicry will not explain the resemblance in every case. Some nervous diseases may closely resemble hysteria. I remember well one case in which hysteria was diagnosed by at least five doctors, including myself, and in which an intracranial sarcoma was discovered post-mortem. Again, hysteria has to be differentiated from malingering and fraudulent shamming. Hysteria is not the same as hypochondriasis; neither is it a form of insanity, though in some cases it may be difficult to draw the line. The term "hysteria," in the sense in which it is now employed, is not, strictly speaking, correct. Sir James Paget suggested "neuromimesis," or "mimicry," and Mr. Holmes uses the word "simulation." Both are good expressions, but neither describes fully the various conditions that are classed together as hysterical.[1] After all, the very indefiniteness of the terms "hysteria" and "hysterical" makes them the most suitable to use, at least until some better expression is invented.

Desiring to make the following remarks as practical and useful as I can, and realising from my own experience that the initial, and I may say main difficulty in cases of hysteria, is the diagnosis, I shall treat my subject mainly from that point of view. Is it not the case that in every instance of hysterical spine, or joint affection, the difficulty is to make sure that there is not present some real organic disease?[2]

Any one who wishes to enjoy a masterly description of the subject should read Sir James Paget's clinical lectures on Mimicry. In what I am now writing I shall follow these lectures somewhat closely, and will refer to them frequently.

In any given case, according to Paget, we have three things to investigate—the etiology, the nervous history and condition of the patient, and the condition of the part affected. He urges that the last is the most important, and most to be relied upon in forming a diagnosis.

Let me suppose, then, a case of hysterical joint affection. The patient comes, or is brought to us, supposed to be suffering from joint disease. There will probably be a history of some injury, or of some mental shock, or the affection may have come on gradually. We may also elicit some information that, taken in relation with the local conditions, may throw light on the case. For instance, an injury may have occurred so long before, that, had actual disease existed all that time, changes would be evident in the joint of a very marked and easily recognised character. The condition of the joint, however, may be quite different. And the want of due relation between time and symptoms may show that no real disease is present.[1]

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[1] Brodie included them all under the term "neuralgia of joints."

[2] Paget says that it is more difficult to diagnose and cure hysterical affections than actual disease.

Valuable evidence may be obtained by knowing that the affection came on after some mental shock, fright, worry, disappointment, debilitating fever (Jolly), overwork, or some such nervous perturbation.

Reliable data may be obtained from the personal and family history. A full treatment of these will be found in the general article (p. 132). I shall merely say here that a neurotic or alcoholic personal and family history will make hysteria more probable, while on the other hand a gouty, rheumatic, syphilitic, or tuberculous history would make one more cautious in coming to such a diagnosis.

According to Paget the patients are mostly females, of nervous temperament, easily fatigued, anæmic, liable to flushes, sensitive to pain, or anæsthetic. They think of themselves constantly and of the part affected; "egotism has its keenest life at and about the supposed seat of disease." They are "content, almost happy, in their afflictions," or they are discontented, and make everyone miserable around them. They are fond of telling everyone of their troubles, and thus court sympathy, for which they have a morbid craving. Will power is deficient in one direction, though some have very strong wills when their interests are concerned. "I cannot" looks like "I will not," but is "I cannot will" (Paget). The family history will disclose a heredity of insanity, epilepsy, intemperance, or some other nervous disorder.

All these points will have been made out while we are examining the affected part, and, if the case be one of simulation or mimicry—a hysterical joint—we will find the following leading characteristics or symptoms present:—

1. *Pain.*—The pain complained of will be in excess of what we usually find in the disease that is simulated. The pain will also be superficial, rather than deep, gentle pressure and pinching the skin causing as much or even more expression of pain than deep, firm pressure. The pain will also vary at different times,[2] may shift from place to place, and may occur in places that we do not usually find painful in actual disease. Pain may be associated with or may alternate with anæsthesia. It seldom, if ever, interferes with sleep.

In joints the forcible approximation of osseous surfaces does not cause pain if carefully performed, and there is no starting at night of the typical kind.

2. *Temperature.*—The general temperature is never raised in the same way as by disease. If there be any rise it will be irregular, capricious, or perhaps fictitious. It is more common to have the general temperature subnormal. If the temperature be above normal the patient should be examined all over. There may be some disease elsewhere causing the rise (Paget).

The same may be said of the local temperature. It is generally lower than normal, the affected part being blue or dead white. If there be any rise of temperature it will be associated with a flushing of the skin and quite temporary.

Paget dwells strongly on the diagnostic importance of the peculiarities of temperature, and points out the risk of being misled by this flushing. He considers that in highly nervous persons any part of the body may flush, just as the face does, from special attention being drawn and directed to it.

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[1] Holmes says "there is no commensurate impairment of health."

[2] Pain may remit, or intermit and recur suddenly (Jolly).

He considers also that a transient gouty condition may give colour and heat to a hysterical joint. These points have to be made out by the surgeon while he is examining the joint—passing the hand over it, gently moving it, closely scrutinising it, and at the same time questioning the patient, both with the object of diverting her attention from the part being examined, and of eliciting information as to the patient's history, constitution, heredity, and other subsidiary symptoms.

In carrying out these manipulations another symptom will come into prominence:—

3. *Rigidity.*—Stiffness or rigidity of the joint may be present to an excessive degree, in the flexed position if the hip or elbow be affected, in the straight position if the knee. On the other hand, but less frequently, the limb may be limp and useless, the patient complaining of weakness rather than of pain—free movement being possible—such as would not occur in disease. Rigidity is more common and is usually excessive, that is to say, greater than is met with in actual disease, all movement being strongly resisted. Sometimes it is possible to see the patient stiffening the limb in anticipation of the surgeon's manipulations. In such cases, if the patient's attention be diverted by conversation, the limb may be gently yet freely moved.

In this connection I may refer to the position which the limb may assume. Paget says, "Subjective intensity of pain brings about the same postures as are assumed instinctively for the relief of pain." He also points out that there may be unwilling or unconscious imitation. This we should always bear in mind.

Barker says that the limb assumes the position commonly observed in disease. This, however, is not always the case.

4. *Swelling.*—Swelling is said to be conspicuous by its absence. My own experience is that there is generally an *appearance* of swelling of the affected joint and also of wasting of the muscles of the limb. But both these conditions are more apparent than real in hysterical affections.

Swelling of a joint is made more apparent by muscle wasting, as in other affections. Careful measurements and comparisons with the other limb will demonstrate that the joint is practically the same as the other. There may be some puffy swelling, but that is generally slight. Wasting of the limb, however, is usually present to a certain extent. This is only what one would expect from the limb not being used as much as the other one. But there is something more that has to be taken into consideration. There seems to be a possible simulation of atrophy, a hysterical shrinkage (if one may use the term) that occurs, and is very apt to mislead. This disappears under the influence of an anæsthetic.

A hysterical limb may, however, be quite plump, with a good layer of subcutaneous fat. Such a state of matters should make one suspicious. But I have, on the other hand, seen cases of tuberculous disease in which there was a fair amount of fat on the affected limb.

Lastly, one may meet with hysterical œdema; that is to say, fictitious œdema that may disappear under an anæsthetic, and is not therefore due to disease. Charcot describes this hysterical œdema. I have myself seen it in a case which I published in *The Hospital* in 1894.

Now let us still suppose that we have our patient before us. We have carefully examined the case, especially locally, tested the pain, the temperature, the rigidity, the apparent swelling and atrophy. If we still have any doubt we should employ a general anæsthetic. There may be some incipient or latent affection, the symptoms of which are very slight, or perhaps masked by the hysterical condition of the patient.

Suppose the patient to be under the influence of chloroform. If the symptoms resembling disease gradually disappear, and as gradually reappear as the patient comes out, then we may presume that the case is hysterical. If, on the other hand, we discover some evidence of disease, such as grating, we can be quite sure that disease is present.

One should not forget that in a hysterical patient there may be great exaggeration of a symptom, such as pain. This is not a case of hysterical joint, but of joint disease in a hysterical patient, and has to be treated as such. We should also remember that a long continuance of hysterical conditions may bring about actual disease. Mr. Holmes says that the influence of the mind on the body is so powerful that it "may lay the foundation of organic disease."

Before leaving this subject of diagnosis let me say that there is one thing, which I cannot call a symptom, very characteristic of hysterical affections, and on which I have very much relied in forming my diagnosis. It cannot be put in one word. It is the presence, more frequently the absence, of some symptom which would not be absent or present in the disease which is being simulated. In other words, the symptoms are not according to type, they are not convincing, they have not the ring of truth about them. One's suspicion is aroused. Something is here that should not be, or something is awanting that ought to be here. Paget says, "The absence of symptoms should be weightier than the presence." Mr. Holmes says that the local symptoms show contradiction or inconsistency. Another way of putting the matter is, in the graphic words of Paget, "There are sensation signs of disease without a single mark of any organic change produced by them." That is to say, you don't find grating, unnatural mobility, osseous thickening, pulpy synovial membrane, actual swelling, or such like conditions which, if present, would prove decidedly diagnostic of disease.[1]

Now, at the risk of repetition, I must put the case again shortly. To reach a correct diagnosis the surgeon must examine the joint (or other part) carefully, with an unbiassed mind, and with the object distinctly before him of *discovering disease if it exists.* If, under these conditions, he is not convinced that disease is present, then he may conclude that the condition is hysterical. If, on the other hand, he finds the slightest trace of disease, or even is unable to say certainly that no disease exists, it is safer to treat the case as one of real disease. Diagnosis should, however, not be uncertain in the vast majority of cases.

I have not yet spoken of the connection between hysterical affections and the sexual functions. In the female you may find an enlarged or sensitive ovary, or the symptoms of which she complains may vary with the menstrual periods.

In the male you may have an undescended or partially descended testis or a phymosis. In either sex there may be the practice of masturbation. Dr. Jolly says that "irritative pathological conditions of the genitals," which he defines as "sexual abstinence or over-irritation," are often present in hysterical patients. These give rise to "diseased reflex actions" on account of the "increased irritability of the sensory system."

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[1] Hysteria may be produced by some local affection. An example was published in the *British Medical Journal*, 28th July 1900, in which the irritant was an ingrowing toe nail. This is not the surgical hysteria of which I am writing. There is no simulation of disease. Any other peripheral irritation might produce the same effect.

This connection between the sexual functions and hysterical affections does not, in my experience, always exist.

HYSTERIA OF THE SPINE.—This subject will be dealt with also under the heads of spinal affections, orthopædics, etc.

I would merely say on the general subject that diagnosis is often very difficult, and that therefore a careful and thorough examination is always necessary. It is not difficult to overlook a slight rigidity of the spine, or a little difference in the length of the lower limbs.

I shall not say anything about lateral curvature as I am sure it will be fully dealt with elsewhere. What I have got to say will be limited to the diagnosis of hysterical conditions from Pott's disease of the spine.

We may further limit our subject to the earliest stage of caries before any prominence has formed, because the advanced stages of this disease cannot be simulated.

In forming a diagnosis the most important symptoms to depend upon are pain and rigidity.

In Pott's disease the pain is produced by firm pressure on the spinous processes of the affected vertebræ, and the same pain is produced by downward pressure on the shoulders or head. In more advanced cases you have the patient supporting the head with his hand, or lifting the weight off his back by placing his hands on the chair or bed when sitting up.

In hysteria, on the other hand, we have the pain (as we have seen) excessive, superficial, varying, and not caused by downward pressure, if this be made carefully and without the patient knowing why it is done.

As regards rigidity there is an equally well-marked distinction. In caries you have this symptom always present. It is made out by causing the patient to flex, extend, and rotate the spine, when some stiffness in movement, or actual fixation of the diseased portion of the neck or back, will be at once made evident.

In hysteria, on the other hand, the back is more frequently limp and weak. Rigidity if present is likely to be spasmodic and of the nature of opisthotonos.

There is one thing that I think should always be borne in mind, viz. that in many persons there is a sensitive area in the spine that has no relation with disease in any way whatever.

Girdle pain is not an infallible proof of caries of the spine. It can be imitated. But as this symptom is not usually an early one in Pott's disease the absence of a projection and of other symptoms will show that the case is one of simulation.

One must remember that severe spinal pain, somewhat hysterical in character, may be caused by a malignant tumour or aneurysm. Neither must it be forgotten that we may have caries occurring in a hysterical subject.

HYSTERICAL AFFECTIONS OF JOINTS.—The diseases which are apt to be simulated are tuberculous disease of the hip and knee, Brodie's abscess, loose cartilage in the knee, and chronic synovitis (of adhesions, contractures, and deformities I will speak afterwards).

The Hip.—The symptoms that will be imitated are pain, lameness, flexion of the joint, lordosis, lateral curvature, flattening of the hip, and adduction with apparent shortening of the limb. As in the case of Pott's disease it is only the early stage that can be simulated.

Let us imagine a case. The patient, who is usually delicate looking and anæmic, limps or is carried in, or we may have to visit her lying in bed. On examining we may find all the symptoms above enumerated apparent. On careful investigation, however, we will find that the pain is, as usual, exaggerated, superficial, and not increased by pressure on the trochanter, heel, or knee. All deformity can be made to disappear under careful and gentle manipulation. The normal movements of the joint may also be obtained if performed cautiously and when the patient's attention is otherwise occupied. If the patient be a female the iliac fossa should be examined for a sensitive ovary, especially if there be well-marked flexion of the hip with lordosis. A very good plan, especially in young persons, is to examine the healthy limb first. In doing this the forcible flexion test for hip disease may be employed with advantage. If the healthy limb can be thoroughly flexed on to the abdomen without raising the affected limb, the latter cannot be diseased. The patient may then be made to stand and walk, and to stand on one leg—first on the sound one, and then on the affected one.

If the patient be a boy he should be examined for phymosis and hernia. I have had two cases of complete cure after circumcision. If there should still be some doubt the patient should be placed under the influence of an anæsthetic, and the conditions of the joint carefully and thoroughly examined. There are reasons why an examination under an anæsthetic may be specially necessary in the case of females.

The Knee.—Tuberculous disease or chronic synovitis are most frequently simulated. The patient limps badly, or refuses to use the limb at all. There may be the history of an injury—perhaps months or even years before. Pain is said to be very severe especially on movement. Examination should be made with both limbs bare so that the surgeon may be able to compare them. At first a difference between the two may be apparent, but closer examination will show this to be slight if at all.

On looking for painful spots these will be found to be, as usual, superficial, variable, and uncertain. By this the possibility of a Brodie's abscess may be often at once eliminated. Anæsthetic areas also may be made out. It will generally be found that the pain does not prevent sleep, and that there is no starting at night. This last symptom may be simulated if the patient has heard or read about it. The temperature of the joint will be normal or subnormal, the skin being sometimes blue or dead white in appearance. The joint may seem to be swollen, but there will be no fluctuation, or thickening of the synovial membrane. The bones will be normal and the patella will be movable, but not floating. The relation of the bones to one another will also be normal. There may be some "puffiness" or even œdema, and perhaps a transient redness—as has already been pointed out.

If the patient be otherwise well nourished a comfortable layer of subcutaneous fat will be easily made out.

On the other hand, atrophy of the limb may exist to a certain extent from want of use, and may be increased by what I have ventured to call "hysterical shrinkage." Careful measurements will, however, demonstrate that the joint is not enlarged, and that the muscle wasting is more apparent than real. The position which the limb usually adopts is straight, but the joint may be flexed. The limb is usually rigid with the muscles firmly contracted so as to prevent motion, which is said to be very painful. If the surgeon manipulate carefully, however, he may be able to satisfy himself that mobility is present and is perfectly normal and painless.

In some hysterical cases, instead of pain with rigidity, the patient complains of weakness. She does not use the limb, and, in consequence, there may be considerable atrophy. One very important point, on which Paget lays considerable stress, is, that the apparent disease is not so advanced as would be the case had actual disease existed for the period during which the patient has complained.

Sometimes swelling and redness may be caused by irritating applications. If this be done fraudulently it can generally be discovered by investigation.

One must be careful not to overlook some chronic or subacute affection, such as gout or rheumatism.

The symptoms of a loose body or cartilage in the knee joint may be simulated. Generally, however, the pain complained of is excessive and uncertain in character.

Lastly, under an anæsthetic all symptoms disappear, and as the consciousness returns so do the symptoms. This generally settles the diagnosis.

HYSTERICAL AFFECTIONS OF MUSCLES.—These are often pure examples of mimicry. The patient sees someone affected with the genuine disease, and, consciously or unconsciously, voluntarily or involuntarily, imitates the movements. As examples I shall refer to only two affections, spasmodic wry-neck and squint.

Wry-neck, if hysterical, comes on suddenly, varies in degree, may intermit, and may pass from the one side to the other. It disappears entirely during sleep. It may be connected with intestinal irritation. It tends to disappear and may do so suddenly.

This will be more fully discussed under "Wry-neck" and "Spasmodic Affections of Muscle." (See also "Strabismus.")

HYSTERICAL TUMOURS.—Abdominal phantom tumours, as they are called, seldom come into the hands of the surgeon. They generally go to the physician and obstetrician. I have little to say of them beyond that they disappear when the patient is placed under the influence of an anæsthetic.

Mamma.—Painful affections of the breast are sometimes supposed by patients to be malignant tumours. If the non-existence of a tumour be made out the patient is generally much comforted. If, however, she be hysterical, she may prefer her imaginary tumour and may disbelieve the surgeon's comforting assurances. This is a very characteristic symptom. In regard to such cases Paget says that we must judge by what we feel, and not by what the patient feels or says she feels.

The pain complained of in hysterical breast is, as usual, excessive and varying; sometimes passing from one breast to another. I should like to say in this connection, that in my experience the removal of the breast for neuralgia (hysterical?) when no actual disease can be made out is most unsatisfactory. The patient may be better for a time, but the pain returns in the other breast, and if that be removed, as I have seen done, the pain is transferred, after a time, to some other part.

HYSTERICAL INTESTINAL OBSTRUCTION.—Hysterical subjects are well known to be prone to constipation. They are neglectful of the calls of nature, and are sometimes even dirty in their habits. Under such circumstances it is possible for the bowels to become torpid, and loaded with fæces to such an extent as to cause obstruction. Such cases differ in no respect from ordinary examples of fæculent obstruction as regards either symptoms or treatment.

But there is another class of cases in which obstruction seems to occur from much the same cause as retention of urine in hysterical females. The patient seems to exercise an inhibitory influence over the intestinal peristalsis, with the result that there is constipation and also sickness. In my experience the diagnostic point is that the sickness has not the well-known characteristics of obstruction vomiting, and is never fæculent. On the other hand a powerful purgative, which would be dangerous in a real case of obstruction, is usually quite successful in overcoming the patient's inhibition of her peristalsis, and in getting rid of the obstruction.

Massage has also been successful in such cases.

HYSTERICAL CONTRACTURES.—While these are described in the general article (p. 120) it may be advisable to refer briefly to their surgical bearings.

Such cases, in my experience, are not usually purely hysterical. There is the history of an injury or of some joint affection. The case has, however, become hysterical because the dominant factor standing in the way of recovery is the unwillingness of the patient to make the necessary efforts, or to use the necessary means for recovery.

Let me put the matter another way. In making our diagnosis we have to settle whether the case is one of pure hysteria, or of hysteria affecting a joint or group of muscles that once were inflamed, or of hysteria grafted on to a pathological condition the result of some previous affection.

To arrive at a correct diagnosis in such cases it is generally advisable to place the patient under the influence of an anæsthetic.

Before deciding we must investigate into all possible causes of contracture, such as joint disease, nervous affections, muscle affections and adhesions.

We must not forget that permanent muscle changes may result from long-continued hysterical contractures. Jolly says that contractures may occur after a hysterical convulsion.

Under an anæsthetic any adhesions will usually be both diagnosed and broken down easily.

TREATMENT.—What I have to say on the subject from the surgical point of view must be necessarily very brief. Paget says that we have three things to treat—the constitution, the nervous condition, and the local symptoms. As regards the first, there is a general consensus of opinion against the employment of narcotics and alcohol. Such pathological conditions as gout, rheumatism, syphilis, struma, anæmia, and oxaluria must be treated *secundum artem.* The patient should have good food, fresh air, change of scene and climate, warmth, and tonics judiciously selected. Sleep should be procured naturally, and for this purpose exercise, especially in the open air, is advisable.

The nervous condition should be treated by moral means. The will should be educated. There should be no threats or forcing (Jolly). And the patient should be separated from her friends.

Hypnotic suggestion has been tried and has succeeded in some cases. I have seen it signally fail.

Now with regard to the treatment of the local symptoms, seeing that these, in hysterical cases, are imaginary and fictitious, their treatment must necessarily be largely moral and through the nervous system.

Holmes tells us to commence with the removal of all possible irritants. These are not always easily discovered. We must investigate the digestive, nervous, circulatory, and sexual systems.

In regard to strictly local applications opinions vary. Of such simple things as heat and cold, we have Paget, on the one hand, stating that warmth is necessary and cold objectionable, while Jolly holds the converse opinion. Probably the question really is, which would have the greater moral effect in any given case. Galvanism also should be employed according to its moral influence. Counter-irritation is often very useful and is best applied lightly and frequently; moral influence again being desired mainly.

Massage has great possibilities, employed both on the Weir-Mitchell principle and locally to a joint or back. Massage should always be applied and administered under the advice and supervision of a doctor.

Surgical operations are not, in my opinion, advisable. They may be temporarily useful in removing a painful part (such as the mamma or testicle), or by leading the patient to believe that some disease has been removed or relieved (moral influence); but the ultimate result is unsatisfactory. The pain, which has been temporarily relieved, recurs in some other part. In a word, the real disease, the hysteria, is not cured, and cannot be cured by such means. There is another kind of surgical operation which may under exceptional circumstances be desired, viz. when a definite diagnosis cannot be arrived at.

Under these circumstances an exploratory incision may be thought of. Personally I do not approve of such operations. To begin with they are an admission of failure. It is said that the slight operation can do no harm, and that the moral effect may be very considerable, the patient being led to believe that a curative operation has been performed. As I have already said, the local effect soon passes of, and the patient finds out that she has been deceived, and the moral effect is unfavourable. On the other hand, it is said that if no disease be found the patient will be convinced on being told this. That is not my experience. I have twice made exploratory incisions (under protest), and in both instances the moral effect on the patient was nil.

If the case be a hysterical one the treatment must be directed to the hysteria. Too much attention may be directed towards the supposed affected part by treatment being applied there. Paget dwells strongly and forcibly on this.

I will, in conclusion, repeat that diagnosis is the main thing. If the case be hysterical, treat the hysteria. If, on the other hand, you have any suspicion that even a trace of disease is present, treat the local affection. It is the less dangerous mistake of the two.

LITERATURE.—Authors referred to: Paget's *Clinical Lectures*, Holme's *System of Surgery*, Brodie's *Diseases of Joints*, Charcot's *Lectures on Nervous Diseases*, Ziemssen's *Cyclopædia of Medicine*, vol. xiv. "Hysteria," by Dr. Jolly; Barker in Holme's *System of Surgery*.

Icterus Neonatorum. *See* New-born Child.

Ichthyosis.

Syn.: *Fish-skin Disease, Xerodermia Ichthyoides.*

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See also Tongue (Ichthyosis Linguæ), Tylosis (Ichthyosis Palmaris et Plantaris). Seborrhœa.

Definition.—A disease present at birth or developing in early infancy, in which the skin is dry, rough, and scaly, with deficiency of normal secretions; or covered partially or generally with horny plates and warty growths. Under this definition are included all the forms of fœtal ichthyosis, the various degrees of ordinary ichthyosis, and the generalised and localised types of ichthyosis hystrix. "Local ichthyosis" or "hyperkeratosis," due to chronic local irritation, nervous diseases or venous stasis, will also be considered. The following conditions are omitted as not being true forms of ichthyosis, viz. "acquired ichthyosis," a disease of advanced life, and due to a form of seborrhœa. "Ichthyosis palmaris et plantaris" and "ichthyosis linguæ" will be found under the headings of Tylosis and Tongue respectively.

ICHTHYOSIS FŒTALIS GRAVIOR.—(SYN.: *Harlequin Fœtus*).—In this rare and most repulsive disease changes in the skin of a most pronounced kind are observed at birth. The epidermis is greatly hypertrophied, hardened, and broken up into scales and plates of different sizes, shapes, and thicknesses, separated by fissures and furrows of varying depth and width. The skin feels hard and leathery, and is of a dirty grey or yellow colour. The fissures and furrows, in contrast to the general colour of the plates, are of a deeper purple or brown. As a result of general contraction of the thickened integument, the mouth gapes, and the nose, as well as the reproductive organs, are practically absent. For the same reason the limbs maintain their intra-uterine position of flexion. The mucous membranes are unaffected.

Such children are almost always born prematurely, while owing to the inability to suck, interference to the functions of the skin, suppuration from pyogenic organisms, visceral and pulmonary congestions, death soon ensues. In one case (Jahn) life was prolonged for nine days.

Etiology.—In about 8 per cent of recorded cases other children in the same family have been born with the same disease.

Pathology.—Various views have been expressed as to the minute changes occurring in the skin.

There is little doubt that the onset of the disease dates from the third or fourth month of fœtal life. Some observers (Simpson, R. Crocker) hold that the condition is an exaggerated form of ordinary ichthyosis, while others (F. Hebra, Bland-Sutton) consider it to be a form of general seborrhœa.

Unna disputes its relation to ichthyosis, and looks upon the malady as a universal congenital hyperkeratosis due to defective nutrition.

ICHTHYOSIS FŒTALIS MITIOR.—Under this name are included the milder cases of fœtal ichthyosis.

Family prevalence as in the grave form is not uncommon, and many cases are prematurely born and die young. In a certain number of cases which reach adult life the condition becomes indistinguishable from one of severe ordinary ichthyosis, and for this reason it may be looked upon as a connecting link between ichthyosis fœtalis gravior and ordinary ichthyosis.

At birth the body is covered with a thin dry pellicle of dark brown or yellowish colour, and after a few days epidermic scales and plates of various sizes are found, with fissures and cracks. The mouth, nose, ears, and genitals are affected as in a harlequin fœtus, but to a less degree.

Pathology.—Caspary found great hypertrophy of all the layers of the epidermis, and a pronounced atrophy of all structural elements of the skin, with the exception of sweat glands, which were in marked evidence.

ICHTHYOSIS SIMPLEX—(SYN.: *I. Vera., Common Ichthyosis*).—Although the more severe forms of this disease were clearly recognised by older observers, Willan was the first to take a comprehensive view of the disease in all its degrees of severity. His original clinical description remains, even at the present day, almost perfect and complete; and his separation of two distinct types—I. simplex and I. cornea—is still generally accepted.

Amongst others Alibert, Rayer, Hebra, Gaskoin, and Erasmus Wilson have greatly helped to advance our knowledge of the disease, while Unna has recently made important observations on its pathological anatomy.

Symptoms.—In the mildest form, to which the names *xerodermia* and *xerosis* are often unnecessarily given, the skin in certain regions, such as the extensor surfaces of the arms and legs, is dry and dirty-looking, with exaggeration of the natural skin lines.

The surface is covered with fine scales, and the hair follicles stand out as innumerable little conical projections, which feel remarkably rough and hard when the hand is passed over them. (*I. anserina vel. keratosis follicularis.*)

Associated with the rough condition of the skin there may be a certain amount of redness, which is liable to become more pronounced in cold weather, or from irritation of any kind. In more severe cases (*I. nitida*) the whole of the surface of the skin is more or less involved, the disease being still more pronounced on the extensor surfaces of the extremities, as in the milder cases. The general appearance of the skin varies with the size, thickness, and colour of the scales.

When the scales are small they desquamate as thin whitish flakes, but when they are large they are of a dirty grey or brown colour, and more adherent. In the latter case the skin is dry, rough, and dirty-looking, the natural lines and furrows are exaggerated, and the skin loses its normal elasticity.

With a further degree of severity the scales are represented by large, thick, polygonal epidermic plates, separated by deep furrows (*I. serpentina, sauridermia*), and adherent to the skin.

The colour in such cases becomes dark brown, green, or even black (*I. nigricans*).

With regard to distribution the disease is most marked on the tips of the elbows, on the fronts of the knees, the extensor surfaces of the extremities, and on the buttocks. The following regions are either affected to a slight extent or are entirely free, viz. the face, where the skin may be rough on the forehead and cheeks, the axillæ, the flexor bends of the elbows and popliteal spaces, the groins, and the genital organs. In nearly all cases the disease is accurately symmetrical in distribution and intensity.

The palms and soles are, in the writer's experience, in most cases hard, dry, and smooth, with exaggeration of the major natural lines and diminution of the minor ones. This opinion is at variance with the statement expressed by Unna and others that the palms and soles are never affected.

The mucous membranes are never involved.

Subjective Symptoms.—Apart from the cutaneous defect subjects of ichthyosis show no other signs of abnormal development and enjoy average good health. Associated with the condition of the skin are certain symptoms, of which a peculiar sensitiveness to changes of temperature, especially to cold, is perhaps the most common. Patients will often volunteer the statement that they "feel the cold terribly."

In many cases there is an entire absence of symptoms, although pruritus, sometimes severe, may give trouble.

Asthma is sometimes associated with the disease, its severity corresponding with the condition of the skin (Jamieson). Acne, boils, and other skin lesions due to pyogenetic organisms are liable to occur, no doubt from the fact that the rough cracked skin is extremely suitable for harbouring micro-organisms.

In addition to these symptoms ichthyotic skin is very liable to be attacked with a moist catarrh or troublesome dermatitis. These inflammatory changes are apt to occur even in the mildest cases from irritation of the skin, exposure to cold winds and heat, or even without any obvious cause.

The recognition of this tendency to inflammation is of the greatest importance both for diagnosis and treatment, for many cases of so-called chronic eczema may be entirely due to this cause, and the underlying condition of ichthyosis may be overlooked.

The persistence of this eczematous condition, starting as it does in early life, may produce a clinical picture closely resembling prurigo. The hair tends to be scanty and lustreless, and is often absent on the trunk and limbs.

The nails are sometimes badly developed, dry, and brittle.

Sweating is diminished in regions where the ichthyosis is pronounced, but may be vicariously increased in parts slightly affected (Aubert). Free sweating in summer gives relief to patients, while wintry weather tends to aggravate any symptoms which may be present. Occasionally, however, temporary improvement occurs in the winter.

Course.—Ichthyosis is generally observed in the first few months of life, and is not often well marked before the third year is reached.

After this age the disease becomes gradually more marked as the child grows older, and reaches its maximum of severity at about the age of 10 years, thereafter persisting throughout life, perhaps improving somewhat in summer, only to relapse again in the winter.

The fact that the disease appears some months after birth, may in part be explained by the action of the amniotic fluid during intra-uterine existence, which prevents the skin from becoming dry; and in addition to this the frequent washing of infants tends to keep the skin soft, and so reduce to a minimum the early evidences of the disease.

When cases are reported as being cured, or as having started after the period of childhood, an error of diagnosis must always be suspected.

Temporary improvement may and often does occur after specific fevers. The disease in all cases must be considered as an incurable one.

Causation.—Heredity appears to be the sole factor in its causation, and ichthyosis may be looked upon as the most hereditary of all skin diseases.

In a large number of cases, relations either in direct or collateral branches of the family are similarly affected.

Although males and females suffer in about equal proportions, there is a remarkable tendency for the disease to be limited to the children of one sex in a family.

As in all other congenital affections, parents will sometimes offer explanations for the appearance of the disease in their children. These are generally of the nature of "maternal impressions."

Ichthyosis is stated to be endemic among two Albanian tribes on the shores of the Adriatic (Tablonowski), and also in Hayti and Paraguay.

Differential Diagnosis.—In ordinary cases there is seldom any difficulty in recognising the nature of the disease. The rough, dirty skin, more marked on the extensor surfaces of the limbs, its onset in infancy, its persistence through life, and family distribution, together form a well-defined clinical picture.

In the first few weeks of life it may be confounded with a condition described by Hallopeau and Watelet as "exfoliation lamelleuse des nouveaux-nés," which is probably identical with the ichthyosis sebacea of Hebra.

In this condition the epitrichial layer of cells, which normally is cast off in the seventh month of fœtal life, persists at birth in the form of a thin, smooth, pellicle like a layer of collodion which is gradually cast off for about two weeks, the infant after that period having a perfectly normal skin.

Morbid Anatomy and Pathology.—Microscopical examination shows the condition of the skin to be due to an exaggerated cornification of the cells of the rete Malpighii with very pronounced thickening of the horny layer.

The stratum granulosum, so conspicuous in sections of normal skin, is entirely absent.

The rete malpighii, according to some observers, is thickened, and its cells abnormally large. Others hold that thinning of this layer is characteristic.

The hair follicles, with the sebaceous glands, are in an atrophic condition, while the sweat glands in most cases appear to be perfectly normal.

The opinion generally held is that the disease is a primary defect of the cornification of epidermic cells, and that consequently it should be looked upon rather as an inherited malformation of the skin than an active morbid process.

Associated with the changes in the epidermis, inflammatory changes are sometimes observed in the true skin, and are considered by most observers to be secondary to the primary disease.

Unna, however, is of an opposite opinion and concluding that the inflammation is primary, he classifies the disease as an "Infectious inflammation."

Treatment.—The disease, it must be remembered, is practically incurable, but treatment, if carefully carried out, may materially alleviate and keep it under control.

The treatment should be directed with a view to remove the dirty and unsightly scaliness, to keep the skin soft with artificial lubricants, and to protect it from any irritation liable to set up the dermatitis which is so apt to occur. Warm baths, to which are added alkalies, borax, bran, or starch, and the use of alkaline soaps, are very suitable for mild cases and especially for children.

For more severe cases in addition to the daily baths, glycerine, diluted to suit the tolerance of the patient, should be frequently rubbed into the skin. By this means the skin becomes soft and smooth, and may be kept in this condition by regular use of lotions containing glycerine, or by the use of lanolin, vaseline, cold cream, or other fatty substances.

When the epidermis is too thick to be removed by these simple means, salicylic acid in the form of an ointment, cream, or paste will be found useful.

To protect the skin from irritation and changes of temperature, flannel should be worn, and its thickness reduced in warm weather. If itching is troublesome the flannel underclothing may be lined with thin washing silk.

Internal treatment is generally supposed to be of little practical use—Besnier, however, recommends frequent small doses of arsenic, while Brocq highly commends cod-liver oil. The writer has seen markedly good results from the use of thyroid extract, quite comparable to those obtained by the same drug in myxœdema. Five grain tabloids once or twice a day given after or during meals are the handiest form of administration. The well-known constitutional effects of the drug must, of course, be carefully looked for, and the treatment stopped or suspended on their manifestation.

ICHTHYOSIS HYSTRIX GRAVIOR. — SYN.: *Porcupine skin, hystricismus, leontiasis hystrix.*

Definition.—A condition arising in early infancy in which the skin is generally, but not universally covered by localised warty epidermic growths or horny masses.

Historical.—No description of this rare disease is complete without reference to the famous Lambert family of which several members appeared in England, France, and Germany, as "porcupine men" at the end of the last and beginning of the present century.

The first case observed in the family was a man in whom the disease appeared when he was about two months old. He had six children, all males, who all developed the disease at the same age. Five of these sons died in childhood, and the remaining one had nine children, two boys and seven girls. The sons again were similarly affected, while the daughters were all unaffected. The disease appeared again in the next generation in a male child of one of these sons.

The disease was therefore present in ten individuals in four generations. In some cases the horny growths were shed at regular intervals.

Clinical.—In the majority of cases the disease appears at the age of about two months, and becomes well marked at an early age.

The general appearance of a case of hystrix may be gathered from the suggestive names given to the condition.

ICHTHYOSIS HYSTRIX MITIOR.—SYN.: *I. hystrix partialis; neuropathic papilloma; nerve nævus; nævus verrucosus, linearis, unius lateris.*

Historical.—The earliest cases were recorded by Thomson in 1829 under the name of "papillary nævus."

Bärensprung, in 1863, pointed out the distribution of the lesions and its similarity to the distribution of Herpes Zoster.

Symptoms.—The condition, although probably always present at birth, may not be noticed until the lesions become more pronounced and papillary at the end of two or three months.

At this age the lesions show their characteristic grouping in lines or streaks, and are more or less soft and brownish in colour.

As age advances the lesions tend to become more prominent, harder, and of a deeper colour. They consist of papillomatous or warty growths either arranged in patches or lines and streaks, which tend to run transversely on the trunk and longitudinally on the limbs.

The skin surrounding the lesions is often deeply pigmented. The presence of these warty growths does not affect the general health. In some cases itching is complained of, while in other cases the diseased areas may become inflamed. If the lesions are in such situations as the axillæ, they may become very vascular and grow rapidly. They are then liable to bleed, fungate, and ulcerate with an offensive discharge.

Mucous membranes are as a rule not affected, but Church describes a case in which the palate, tongue, and inside of the cheek, were affected, and Ryan saw a case in which the palate was alone involved.

In some cases epilepsy and other nervous manifestations are associated with the disease.

Causes.—The sexes are affected in about equal proportions, and there is no evidence either of family prevalence or hereditary transmission.

The distribution often corresponds to some cutaneous nerve, and sometimes follows the deep nerves of the part. In some cases, as pointed out by Phillippson in 1890, the lines and streaks correspond to "Voigt's lines," *i.e.* the lines of intersection between the areas of distribution of the cutaneous nerves. In other cases the distribution appears to correspond to the lymphatics of the part (Heller), or to metameric developmental fissure-lines (Unna).

Pathological Anatomy.—The papillæ of the corium are markedly enlarged, the connective tissue is loose in structure, the elastic fibres are either atrophied or entirely absent, and the arteries enlarged. The stratum corneum is greatly hypertrophied.

Kerato-hyalin and eleïdine are abundant in the granular layer which is well developed. The rete Malpighii is reduced in thickness and deeply pigmented in its lowest layer.

Dilatation of the sweat ducts is a marked feature in many cases, but the sebaceous glands and ducts are normal.

Treatment is generally only necessary when the lesions are unsightly from being situated on the face, neck, or hands, or when the local irritation or inflammation gives much trouble.

For small growths temporary or even permanent relief may be given by using a strong solution of salicylic acid or resorcin in collodion, or by repeated paintings with tincture of iodine.

For larger growths, free removal either with the galvano-cautery or with the knife is to be recommended.

LOCAL HYPERKERATOSIS.—Under this name are considered several conditions in which thickening of the horny layer of the skin occurs in localised patches.

This thickening may be found on any part of the body, but is more commonly seen on the palms of the hand and soles of the feet, a fact no doubt explained by the normal thickness of the epidermis and the liability of these parts to frequent and various forms of irritation.

Callositas.—The most common form of simple hyperplasia of the horny layer is seen in the callosities, which may develop on any part of the body subjected to continuous pressure or irritation.

These callosities frequently occur in the hands of workers in various trades, such as mechanics, shoemakers, carpenters, tailors, and others, and are directly caused by the irritation resulting from the continued use of hand tools. Similar changes occur on the finger tips of stringed instrument players, on the hands of oarsmen, and the hands of metal workers who handle mineral acids.

The callosities vary in position according to the particular kind of tool used, and, as Hebra pointed out, the occupation of a man may often for this reason be ascertained by examination of the hand.

It must not be forgotten, however, that callosities may develop in other regions, such as under badly fitting belts or trusses, and on the feet from ill-fitting boots.

Local Hyperkeratosis associated with general Skin Diseases.—When the palms and soles are attacked in such diseases as eczema, psoriasis, lichen planus, and syphilis, the lesions often occur as localised patches with marked thickening of the horny layer. This hyperkeratosis tends to mask the usual characters of such eruptions in these regions, and in the absence of lesions on other parts of the body it may be difficult to make a positive diagnosis of the condition.

Of these conditions the most pronounced hyperkeratosis is found in the tertiary syphilide localised on the sole of the foot.

Arsenical Keratosis.—This condition is caused by the long-continued use of arsenic, and is usually accompanied by arsenical pigmentation, peripheral neuritis, or other manifestations of chronic arsenical intoxication.

The eruption which generally appears first on the hands, and more rarely on the feet, consists primarily of patches of small warty growths situated round the orifices of the sweat ducts, and subsequently of callosities with a roughened dry condition of the intervening skin. Chronic hyperidrosis is a frequent concomitant, and it may precede the development of the hyperkeratosis by a considerable period. As the lesions enlarge they tend to lose their nodular appearance and become diffused, raised, thickened, rough patches. These changes tend to persist indefinitely even after discontinuance of the drug.

The most important point in connection with arsenical keratosis is its liability to become the seat of epithelioma.

Other forms of Local Hyperkeratosis.—Prolonged hyperidrosis of the palms and soles, common in neurotics, excessive tea drinkers and alcoholics, may lead to considerable horny thickening, the skin becoming smooth with loss of the smaller natural lines. Occasionally considerable pain and tenderness is complained of, and painful cracks and fissures may form.

Local horny thickenings have been observed as a result of venous stasis as a sequel to various nerve injuries, and during the course of tabes dorsalis.

Of the more rare forms of hyperkeratosis may be mentioned the *keratodermia erythematosa symmetrica* of Besnier, in which there arises in early life multiple scaly patches on the palms and soles surrounded by a narrow erythematous zone. The condition is not hereditary like tylosis, and its cause is unknown. It gives rise to no severe symptoms, and persists throughout life.

A somewhat similar affection has been described by Brooke under the name of *erythema keratodes symmetricum.*

A sharply defined erythema appears on the palms and soles, and is followed by a superficial hyperkeratosis with some œdema and tenderness. This condition is readily cured, and appears to be more of the nature of a primary inflammation.

Treatment.—When the epidemic thickening is associated with some general disease the treatment should be modified accordingly.

In cases where the condition is due to chronic irritation or continuous pressure these causes must be removed if cure is to be permanent.

For local treatment the thickened tissue should be softened and pared down with a knife.

For softening the surface the skin may be bathed with hot water and soft soap, and afterwards treated with wet dressings, such as solutions of caustic potash, salicylic acid, or acetic acid. The plaster muslins made by Beiersdorff, of Hamburg, containing salicylic acid, creasote, resorcin, and pyrogallol, if persistently used, may bring about a permanent cure.

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Idiocy. *See* MENTAL DEFICIENCY.

Ileus. *See* INTESTINE.

Immunity.

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INTRODUCTION. — The conception of immunity was first formed when physicians learnt that after recovery from serious diseases, and especially after small-pox, a life-long protection may be secured from like diseases. Jenner then pointed out that enduring protection against a disease can be acquired by undergoing a similar or nearly related disease (as in vaccinia and small-pox), and since 1880 Pasteur's discoveries in regard to various artificial protective inoculations have given us a clearer conception of the nature of immunity. Gradually thereafter arose the questions, which are of both theoretical and practical interest, as to why man is not susceptible to so many parasitic diseases, and as to how the living organism is enabled to withstand the putrefactive bacteria, whether they be those of the intestinal tract or of the external surface of the body, which manifest their destructive power immediately after the death of the whole body or of individual parts. In both these instances, however, we are obviously dealing with other circumstances and conditions than in the case of protective inoculations. It is customary, therefore, to differentiate "natural" or "innate immunity" from "acquired" or "artificially induced immunity." But in order that the subject may be clearly understood it is important that our nomenclature should indicate the existence of this fundamental difference, hence in the following pages the innate condition of immunity will be designated as "natural power of resistance," whilst the term *Immunity* will be reserved exclusively for the specific condition which has either been acquired or has been artificially produced by protective inoculation. In short, specific immunity, on the one hand, is characterised by the presence in the body of specific antitoxins, or, speaking generally, of specific anti-substances which have been formed in the body in consequence of undergoing a specific disease, or by means of specific treatment. On the other hand, the natural power of resistance depends chiefly on the capability of the leucocytes to act either as phagocytes or as elaborators of microbicidal alexins, which are totally different in constitution and mode of action from antitoxins and anti-substances. Again, it should be noted that there are two main types of infective processes: one in which the morbid changes are caused by great multiplication of the virus in certain parts of the body or throughout the blood generally, for example, anthrax, and a second type in which the virus remains localised at the seat of invasion whilst the morbid changes of the body in general are due to the absorption thence of toxic products formed locally, for example, tetanus. We can thus differentiate "pure bacterial infection" from "toxic bacterial infection," and it is therefore only natural, when dealing with specific immunity, that "simple immunity to bacteria" and "immunity to toxin" or "tolerance of poison" should be distinguished from one another. It is obvious that we cannot always draw such hard and fast lines, and many infective processes represent an admixture of the two types. We may regard anthrax, tuberculosis, leprosy, and glanders (malleus) as examples of "pure bacterial infection,"—tetanus, diphtheria, and Asiatic cholera as instances of "toxic infection,"—typhoid fever, plague, influenza, septico-pyæmia, etc. as being "mixed forms." From a practical standpoint immunity to toxin is of more importance than immunity to bacteria, because in toxic infections, for example, diphtheria or tetanus, the transference of antitoxic serum from a specifically treated—actively immunised—animal may confer very marked protection, and may induce a cure if there has been pre-existing infection (*passive* immunisation), whilst in pure bacterial infections such protection cannot be conferred by the blood serum of an actively immunised animal, for instance in anthrax, tuberculosis, etc. In the mixed forms the success attained by passive immunisation, *i.e.* by serum therapeutics, is varied, but usually slight. By active immunisation, *i.e.* by inoculation with killed cultures, however, man may obtain a certain degree of protection from Asiatic cholera and plague (Haffkine).

Another point of practical significance is that the natural power of resistance may be increased by certain means, and that this increase is manifested either throughout the whole body or merely in certain localised parts. In endeavours towards this end medicine touches upon what may be called the question of personal or individual hygiene, which likewise aims at increasing the power of natural resistance against disease.

I. Natural Power of Resistance (Innate Immunity)

A. Natural Resistance to Bacteria.—Each infective agent endangers the various species, varieties, and even races of animals to a different degree, and no infective agent is of the same danger to all. Infective agents have only a limited sphere of action in the animal kingdom, whilst most species, etc. possess a natural power of resistance against them. The human subject, for example, appears insusceptible to cattle plague, fowl cholera, swine erysipelas, etc., whilst all animals are resistant to the micrococcus of gonorrhœa, to scarlatina, measles, influenza, and so on. Even closely allied varieties may manifest great differences in susceptibility; for example, the field-mouse (*Arvicola arvalis*) is very susceptible to glanders and tuberculosis, whilst the common house mouse and white mouse are resistant. The marmot (*Spermophilus guttatus*), which is indigenous to southern Russia, possesses a remarkable susceptibility to glanders. Even racial differences may play an important part. Algerian sheep, for example, are much less susceptible to anthrax than are the races of European sheep. Similarly, certain breeds of swine (Yorkshire hogs) are decidedly more resistant to swine erysipelas than are other breeds. Differences are also found among the races of men. Negroes are noted for their remarkable powers of resistance to yellow fever, and in a lesser degree to malaria, yet they quickly sicken of and succumb to tuberculosis and small-pox.

In identical species and races, age, the state of nutrition, and other circumstances are important factors. Young individuals are in general less resistant. Young pigeons, for instance, may be infected with anthrax, whilst the older birds can be infected only when their physical state has been altered by prolonged fasting. The same holds true for anthrax in dogs, their power of resistance being reduced if water is withheld from them. Tesser and K. Müller by feeding rats exclusively on a bread diet succeeded in rendering them more susceptible to anthrax than when they are kept on a meat diet, and Charrin and Roger obtained similar results by fatigue induced by making rats run in a revolving wheel. Pasteur's experiments on fowls and pigeons are celebrated. The birds' normal temperature (42° C.) was lowered by immersion in cool water, and the power of resistance against anthrax was thus abolished. Many animal experiments undertaken by Wagner, Lode, and others prove that the natural power of resistance to various infections can be lowered by artificial cooling. The injection of chloral, antipyrin, or phloridzin also diminishes the power of resistance. In the human subject, besides such factors, for example, cold, as already mentioned, conditions of psychical depression exert an important influence in lowering the power of resistance, as in tuberculosis. Again, the power of resistance which is commonly present may be suspended by local injury to certain organs, or by local factors which from the outset favour the multiplication of the infective agent, or its transference into the blood. Severe bruising of the periosteum, for example, not infrequently induces pyæmia, in consequence of the staphylococcus settling down in the contused periosteum. Boils on the lips are known to be very dangerous, because the staphylococci gain access to the blood with unusual rapidity from the tissue of the cheeks, which are rich in lymphatics.

B. Causes of Natural Resistance to Bacteria

We must, in the first place, mention the *external* protective appliances which retard the penetration of infective agents into the body. Provided there be no separation of continuity the external skin affords protection, which is, however, insufficient on inunction of the infective agent with mechanical pressure. There is a second line of defence in the lymphatic glands, which mechanically retain the infective agents taken from the lymphatic vessels, and the organism is thus afforded time to rid itself of the virus by its further protective powers (*vide infra*). Intact mucous membranes are, for the most part, impervious to infective agents. Large quantities of virulent anthrax spores, for example, are required to infect guinea-pigs or mice by the intestinal canal, whilst very small quantities suffice to infect either subcutaneously or by the lungs if the infective material be finely pulverised. Other truly protective appliances of the body are the acid reaction of the normal stomach contents, the bactericidal properties of the saliva, of the vaginal and urinary secretions, and so on.

It is evident that the essential cause of natural resistance to bacteria does not consist in these external protective appliances, but in internal and somewhat complex conditions of the bodily organism of which we have as yet only an incomplete knowledge; yet the facts which we do know are from the practical aspect those of the greatest importance. It was formerly thought that infective agents could not multiply at all in a resistant organism on account of unsuitable food-supply. Such a conception is, as a rule, incorrect, for the bacteria usually multiply at first—for instance, in superficial wounds—yet after a time the multiplication ceases and recovery ensues. Hence the problem is as follows:—What are the factors which effect this change? by what means is the multiplication of bacteria inhibited whilst there is manifestly suitable pabulum for them in the tissues? The change can only be induced by the direct action on the bacterium of the prophylactic appliances of the body two of which are known, viz. phagocytes and alexins.

1. *Phagocytes,* according to Metschnikoff (phagocytic theory since 1884), are in part wandering and in part fixed tissue cells, which, by throwing out protoplasmic processes, are capable of developing, of then digesting, and of thus removing infective agents which have penetrated into the body. The chief wandering phagocytes are the mono- and polymorpho-nuclear leucocytes (with the exception of the small lymphocytes and of Ehrlich's mast-cells), and the so-called wandering tissue cells; whereas many endothelial cells, especially Kupfer's star cells, the cells of the splenic pulp and of bone-marrow, sometimes also connective tissue cells, and even nerve and muscle cells, act as fixed phagocytes.

The first decisive observations were made by Metschnikoff in the fungus disease of the daphnia, a species of water-flea, where the fungus spores are devoured by the leucocytes of the daphnia, and a cure thereby effected. Metschnikoff afterwards detected a similar process in frogs affected with anthrax, and more especially in them did he observe the death of anthrax bacilli inside the leucocytes of the frog, a process associated with swelling of and degenerative changes in the bacilli. By numerous observations Metschnikoff later on proved that phagocytosis may be perceived in the course of all infective processes, and more especially if the animal be resistant, *i.e.* if the process end in recovery. If micro-organisms are brought into direct contact with the phagocytes of a resistant organism—for example, if injected into the blood-vessels—they are then taken up by wandering or fixed phagocytes. Should the micro-organisms reach a part where there are few or no phagocytes, the subcutaneous tissue for instance, numerous leucocytes migrate towards the affected spot, and in vertebrates this process is facilitated by the inflammatory emigration. Thus the local deposition of bacteria induces a local reaction which indicates the inflammatory process. The cause of this immigration consists, as proved by Leber, Massart, Bordet, and Buchner, in chemiotaxis of leucocytes, *i.e.* in the capability of their migrations being affected by the attractive influences of certain chemical substances. We make a distinction between positive chemiotaxis (attraction) and negative chemiotaxis (repulsion). Buchner showed that dead bacteria, the proteid constituents of bacterial cells (bacterial proteins), and, lastly, certain closely allied chemical substances, such as vegetable casein, are positively chemiotactic or attractive. The toxins of many virulent bacteria have a negative chemiotactic or repellent power, so that they are avoided by the phagocytes. Bacterial spores may also be engulfed by phagocytes, and even though not killed will yet be prevented from germinating. Metschnikoff has also proved that not only living but also virulent infective agents may be engulfed by phagocytes. Yet, when infective agents which have been enclosed in collodion sacs, and thus protected from phagocytes, are introduced into the tissues, they retain their vitality and virulence a long time, even though the animal be resistant. In natural resistance to bacteria phagocytosis is, according to Metschnikoff, developed to an exceptional degree, and is of such constant and regular occurrence that we may often foretell from the degree of phagocytosis whether or no the animal experimented on will gain the victory over the micro-organisms.

2. Although the observations of Metschnikoff are undoubtedly correct, yet the "cellular theory," *i.e.* the phagocytic theory, cannot be wholly and unreservedly accepted, because the fluids of the animal body contain not only phagocytes, but also bactericidal substances in a soluble form, which are termed alexins, and which are likewise concerned with the destruction of bacteria. Von Fodor was the first to show that freshly-drawn rabbit's blood can destroy anthrax bacteria—a fact confirmed by Nuttall and Flügge—and he also proved that not only defibrinated blood, but also pericardial fluid and aqueous humour of dogs and rabbits, is bactericidal, and that the

blood loses its activity on being heated to 55° C. Buchner, in conjunction with Fr. Voit, Sittmann, and Orthenberger, found that completely cell-free blood plasma or serum has a bactericidal action which is lost at 55° C., but is not altered by freezing and subsequent thawing. Fresh blood, however, when frozen and afterwards thawed loses its bactericidal power, not because the active substances are destroyed, but because the red blood corpuscles are dissolved, whereby the blood becomes a medium so suitable for bacteria that its bactericidal action is compensated for, just as weak antiseptics are if bacteria be well nourished. The destruction of bacteria in serum, however, cannot be referred to lack of nutriment, since serum acts as a nutrient medium after being heated to 55° C. Behring, working with rats which are moderately resistant to anthrax, had demonstrated the destructive action of their blood on anthrax bacilli, and believed that it was to be ascribed to an organic base. Buchner was the first to prove that the protective substances contained in the serum (alexins) are of proteid nature and very unstable.

The proteid nature of these substances is indicated by the proportion of salts contained in the serum. Withdrawal of the mineral salts from the serum by dialysis suspends its bactericidal activity, which is restored, however, by readdition of the salts. But the bactericidal power is not due to the salts alone, for in that case it would be maintained after serum had been heated to 55° C. The action of the salts is consequently an indirect one; it is only when they have entered into loose combination with the proteid alexins that the functional power of the latter can be displayed, just as the functions of the cells and organs of the general body are dependent on their containing a normal amount of mineral salts. Moreover, the alexins can be precipitated along with the other proteid substances of the serum, can be dried and again dissolved without losing their activity. Yet it has hitherto been impossible to truly isolate the alexins because of their instability. They retain their activity for a considerable time in frozen serum, but lose it sooner or later at higher temperature or under the influence of light and oxygen. The alexins of different animal species have different degrees of activity, those of the human serum are very actively bactericidal. The degree of bactericidal activity, however, is largely dependent on the nature of the bacterium employed, and on the relative proportion of serum to the number of bacteria contained therein. A given quantity of serum can only destroy a certain number of bacteria, for the alexins themselves are destroyed or used up by contact with bacteria. This is the explanation of the increased danger to which the body is exposed when the infective agents are numerous. It is almost immaterial whether we inoculate a dead sterile nutrient medium with a few or with milliards of germs of one kind; in both instances they multiply until the nutrient medium is all consumed. In the living body, however, the results will be very different, for resistance is more likely to be maintained and recovery to occur if only a few germs be employed. The action of alexins on bacteria is probably a proteolytic one on the bacterial cell plasma. We may therefore assume that the alexins are deleterious to other cells foreign to the body, *e.g.* they dissolve the hæmoglobin out of the red blood corpuscles of a different animal species (hæmolytic action, *vide infra*). An explanation of natural resistance to bacteria by such action of alexins as just described would constitute a "humoral theory" in contradistinction to the cellular or phagocytic one. But the "humoral theory," too, cannot be accepted *in toto*. The real explanation of the problem lies midway between these two antagonistic theories, for, according to all recent investigation, the alexins are mainly derived from the leucocytes.

3. The leucocytes, as is claimed in the cellular theory, must be regarded as the chief cause of natural resistance to bacteria, but not merely because they are phagocytes devouring and digesting bacteria, but also because they produce alexins. The great bactericidal power of exudations containing leucocytes is due to alexins. It was first proved that the bactericidal action of blood and artificial exudations was proportionate to their richness in leucocytes (Denys, van de Velde, Havet, and Kaisin). But the notion

still prevailed that the leucocytes acted as phagocytes, until Hankin and Kanthack indicated that the alexins were a direct product of the eosinophile leucocytes. The experimental proof thereof was afterwards afforded by Buchner and Hahn as follows:—

> Exudations which are rich in leucocytes, yet free of bacteria, are obtained by the injection of sterilised emulsion of wheat-gluten into the pleural cavities of rabbits and dogs, and such an exudation is more markedly bactericidal than the blood and serum of the same animal. This increase of bactericidal power does not depend on phagocytosis, for it is maintained after freezing and then thawing the exudation—a procedure which kills the leucocytes of a warm-blooded animal, but does not destroy the alexins. This was afterwards confirmed by Schattenfroh and Jacob; and Bail and Bordet, working in Metschnikoff's laboratory, proved by a series of experiments that bactericidal substances are supplied to the serum by the leucocytes. Metschnikoff accordingly admits that alexins may be produced by leucocytes, but conceives that they only pass into the serum on the death of the phagocytes ("Phagolysis"), such as occurs in abundance after the withdrawal of blood or after its injection into serous cavities. He says there are no alexins in the normal tissues and the blood within the body because living leucocytes, in his opinion, do not secrete any alexins. Buchner, Laschtschenko, and others dispute this assertion. Whether the question be finally decided in favour of one or other view, yet in either case we would have to conclude that, at the commencement of every infective process, as soon as the normal conditions are altered by the presence of bacteria, phagolysis occurs, *i.e.* death of the phagocytes, whereby alexins get into the locally exuded fluid. But when once there, they must manifest their bactericidal action, and the primary injury to the vitality of the micro-organisms is always therefore due to alexins. This injury is not detectable microscopically, but in consequence thereof the secondary process which may be seen under the microscope, *i.e.* the devouring of the bacteria by phagocytes, is rendered much easier. The detection of virulent bacteria in the interior of phagocytes is no proof against a primary antecedent injury induced by alexins, for the injury may possibly consist merely in a momentary suspension of the vital chemical activity of the micro-organism—a condition of latency, as in bacteria kept at a temperature below 0° C. for example—whereby the elaboration of toxin, and therefore negative chemiotaxis, is momentarily suppressed, whilst positive chemiotaxis due to excretion from the bacterial cell of traces of its proteid constituents alone continues in force. Should the phagocytes afterwards succumb to any cause, *e.g.* to one experimentally induced, the engulfed bacteria may then renew their vital activity, may multiply, and show undiminished virulence.

A view similar to this had formerly been advanced by Bouchard, and such a theory is, it now appears, the most correct. In contradistinction to the phagocytic and humoral theories it may be termed the "alexocyte theory," for the cells by means of their alexins effect partly an intra- and partly an extra-cellular destruction of bacteria which have invaded the body. Alexins, as products of leucocytes, appear to be related to those histolytic enzymes of the latter (Leber), which cause tissue softening in abscesses, and especially in cold abscesses. The proteolytic action of leucocytes can be experimentally demonstrated by the introduction of little cubes of coagulated egg albumin into the subcutaneous tissue of rabbits; the albumin after a time is seen to be softened and digested. The absorption of catgut in completely aseptic wounds is another instance of the proteolytic action of leucocytes, an action which is in keeping with their fundamental character as absorptive cells. Chemiotactic attraction of leucocytes is not only effected by bacteria and bacterial products, but also by vegetable casein, and by all abnormal animal albumins (Buchner). The leucocytes are therefore not merely combative, but also absorptive cells. On this account the alexins, which must be regarded as proteolytic enzymes derived from leucocytes, cannot be considered as formed for the purpose of affording protection against bacteria, but must be regarded as an appliance common to and indispensable for every animal organism.

They normally effect the dissolution of organised substances, yet their action may also be directed against substances and cells which may have penetrated as foreign bodies, whether they be the red blood corpuscles of a foreign species, bacteria, or other microscopic fungi.

C. Increase of Natural Resistance to Bacteria

This may occur in two forms, either generalised throughout the whole organism or locally in particular areas or organs.

1. General increase of natural resistance to bacteria is effected by such agents as induce general hyperleucocytosis, for if the blood contains an excess of leucocytes it possesses increased bactericidal power, unless the leucocytosis be a pathological one, as, for example, in leucocythæmia. Hyperleucocytosis can be effected by various means, but their action is always that of increasing the preventive powers of the body. The following preparations have been hitherto successfully employed subcutaneously:—deutero-albumose prepared by digestion (Matthes and Krehl), organic extracts, spermin, and substances allied to albumose (Goldscheider and Jakob), yeast nuclein and nucleic acid obtained from yeast (Vaughan, M. Hahn), papayotin (Pawlowsky), emulsin, diastase (Hildebrandt), and pilocarpin (Loewy and Richter). Success may also be attained in the human subject (M. Hahn), and according to the results recorded in America the above-mentioned products obtained from yeasts are those whose administration is likely to be the most successful.

Similar effects can be obtained by the use of bacterial proteins—substances extracted from the cells of various forms of bacteria, because they likewise induce hyperleucocytosis (Buchner). The older tuberculin of R. Koch (1891) belongs to this class of substances, and is to be regarded as essentially the protein of the tubercle bacillus. The specific action which Koch supposed this tuberculin to possess is a very limited one. It induces hyperleucocytosis, however, and acts as an irritant on all the tissues of the body, but by careful dosage the irritation amounts to real inflammation with accumulation of leucocytes only in diseased parts where there is some pre-existing irritation.

As bacterial protein is contained in the bacterial cells, it is only natural that hyperleucocytosis and increase of resistance should be induced by the mere use of killed cultures of various bacteria. It was experimentally shown that subcutaneous injection of killed cultures of the pneumo-bacillus may effect a cure from anthrax (Buchner), and that in typhoid fever subcutaneous injection of sterilised cultures of the typhoid bacillus or B. pyocyaneus may sometimes be beneficial, and may considerably shorten the course of the fever (E. Fränkel, Rumpf). Living bacteria of various kinds may consequently be introduced into the animal body; they perish there, and excreting protein they induce hyperleucocytosis and increase the resisting power. Thus rabbits, after simultaneous infection with anthrax bacilli and non-lethal streptococci of erysipelas, recover from anthrax (Emmerich). A similar recovery of rabbits from anthrax can be effected by means of B. pyocyaneus (Bouchard) or various other bacteria (Pawlowsky). For the same reason a severe intercurrent yet not fatal attack of erysipelas may induce recovery from pre-existing pulmonary tuberculosis (Schäfer, Waibel, Macnamara, Thornton, etc.).

The injection of various apparently absolutely indifferent fluids, for example, physiological saline solution, bouillon, etc., into the peritoneal cavity of guinea-pigs may be followed by a temporary increase of natural resistance, which may even simulate a specific immunisation. Cinnamic acid is an agent exciting general hyperleucocytosis. Landerer has for several years successfully treated pulmonary tuberculosis in man by the use of intravenous emulsions of cinnamic acid. The fine particles of cinnamic acid circulating in the blood are deposited in those parts of the body where there are morbid tissue changes, hence in the tissue around tubercular foci. The results of the chronic inflammation in this instance are dilatation of capillaries, œdema and accumulation of leucocytes; at a later stage a thick wall of leucocytes is formed, there is subsequent formation of connective tissue, and lastly cicatrisation.

Finally, we may note that a general increase of the natural power of resistance to bacteria may undoubtedly be affected by general dietetic and hygienic measures, and the most convincing proof of this is afforded by the successful dietetic treatment of pulmonary tuberculosis at the present day. In this instance nutriment suitable both in quality and quantity, suitable alternation in the use of the different organs, bodily activity, pure air, and all strengthening measures are of great importance. We may note more particularly that general hydrotherapeutic measures which attract great numbers of the leucocytes towards the skin may undoubtedly be followed by general hyperleucocytosis. The same is obviously true in the case of sun and light-baths, for there is no doubt that they too attract leucocytes towards the skin.

2. *Local* increase of natural resistance to bacteria is most easily effected by the artificial increase of blood-supply to the part in question. The resistant power of a part may possibly be increased because it is better nourished by blood. Yet the blood must not be regarded solely as a means of transport for nutriment, for it has also, in proportion to the leucocytes and alexins it contains, an important absorptive action on diseased structures, and also, of course, against their causal agents. The experience gained by increasing the blood-supply to diseased parts of the body, such as the limbs, entirely corroborates this view. The most important results in this respect have been obtained by Bier, who was the pioneer of the successful treatment of tuberculosis of joints and bones by means of chronic venous congestion. The following methods of inducing increased blood-supply have been employed:—

(*a*) Venous congestion of a limb by means of elastic compression. This method may be beneficial not only in tuberculosis, but also in gonorrhœal infection of joints, and in acute and chronic articular rheumatism.

(*b*) Arterial hyperæmia is best induced by hot air (100°-150° C. with a special apparatus); for articular rheumatism and arthritis deformans.

(*c*) Mixed hyperæmia induced by Bier's suction apparatus; for chronic articular rheumatism.

(*d*) Increased arterial flow without evident external hyperæmia is caused by the permanent application of alcoholic bandages.

Such bandages were first employed by Salzwedel in cellulitis, lymphangitis, whitlows, boils, mastitis, etc., and with splendid results. In many cases an excellent recovery from tuberculosis affecting bones or joints may follow the permanent use of alcoholic bandages. This action cannot be due, as was supposed, to any direct disinfection by means of the alcohol, for alcohol cannot penetrate deeply into the tissues, and moreover the presence in the tissues of any direct chemical disinfectant is well known to be favourable to bacteria, which, because of their protective membranes, are always more resistant to antiseptics than are the tissue elements. Alcohol, however, when locally applied has, more than any other chemical agent, the direct power of dilating blood-vessels and especially the arteries. In limbs which are enveloped in alcoholic bandages the effect is seen in local increase of arterial pressure, proving that the action of the alcohol is distributed throughout the whole limb. This is the explanation of the beneficial change which occurs so rapidly in cellulitis, for example, after the application of alcoholic bandages.[1]

D. NATURAL RESISTANCE TO POISON

A certain degree of knowledge concerning innate immunity to poison existed even in ancient times. Lucanus, in the Ninth Ode of *De Bello civili*, mentions that the Psylli, an African tribe, were able to withstand the bite of the asp found in their country. They also understood how to cure strangers bitten by snakes, the treatment mainly consisting in sucking the wound and rubbing in abundance of saliva. It is therefore interesting to note that, according to Wehrmann (*Annal. de l'Institut Pasteur*, 1898), ptyalin has a definite antitoxic action against snake poison.

Ages ago it was known that snake venom is comparatively harmless when in the stomach, and such a "regional" resistance to poison (Behring) is found to be very common; rabbits, for example, are resistant to morphia when administered subcutaneously, intravenously, or by the stomach, but not so to intra-cerebral injections (Roux and Borrel). Similarly with tetanus toxin in the case of fowls; they exhibit considerable resistance to this deadly toxin if it be administered by the usual subcutaneous method, yet are killed, even by small doses, if the injection be intra-cerebral.

Those animals, nevertheless, which are insusceptible to a toxin administered by the usual methods, *i.e.* subcutaneously or intravenously, must be denoted as "resistant to the poison." Now there are differences in the degree of resistance, and two different animal species almost invariably exhibit a different degree of susceptibility to a poison. Knorr, for example, ascertained that the following quantities of the same tetanus toxin are required to produce a lethal effect for each gramme of body-weight:—In the horse 1 unit of toxin, in the guinea-pig 2 units, in the goat 4 units, in the mouse 13 units, in the rabbit 2000 units, in the fowl 200,000 units. This table applies, as already mentioned, only to subcutaneous and intravenous, but not to intra-cerebral injection. It has long been known that the hedgehog, the pig, the ichneumon, and many snakes are very resistant to snake venom, and that scorpions are resistant to their own poison. Rats are very resistant to diphtheria toxin, and mice are but little susceptible. Crocodiles are very resistant to many bacterial poisons, and especially to tetanus toxin, being susceptible, however, to diphtheria toxin. The supposition that resistance to poison is due in all these instances to the presence of antitoxins in the animal species in question is on the whole erroneous (Vaillard); such a condition occurs only in exceptional cases, for instance, in scorpions (Metschnikoff). After the injection of tetanus toxin into turtles their blood remains poisonous for several months, *i.e.* capable of inducing tetanus, although the animals themselves are absolutely resistant.

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[1] The alcoholic bandage consists of eight layers of gauze bandage which has been dipped and wrung out in 95 per cent alcohol (60 per cent alcohol is rarely employed) and applied directly to the skin. Over this is placed a layer of cotton wool, and then gutta-percha tissue, which is often perforated. To ensure success it is important that the bandage should cover a large area, for example, in cellulitis of the hand the whole forearm should also be bandaged. The arterial dilatation is due to the cutaneous irritation associated with the dehydrating action of the alcohol, hence propyl-alcohol is more efficacious than ethyl-alcohol, but the use of the former is not advisable, because of its unfavourable by-effects. One result of the locally increased arterial pressure is the disappearance of œdema, and alcoholic bandages are therefore beneficial in non-inflammatory swellings. Lastly, alcohol is of great benefit in caries of the teeth. They should be cleaned twice daily with a tooth-brush which has been well moistened in water, and been then dipped in alcohol, 45 per cent or weaker. After three or four weeks' treatment the caries ceases because of the increased arterial blood-supply to the pulp.

This is accounted for by the absence in this particular species of animal of cells and groups of cells susceptible to poison.

The existence in the different animal species of these fundamental differences in regard to resistance to poison must be taken into consideration in attempting to explain general natural power of resistance. There are cases in which it is only because of the pre-existing resistance to poison that the phagocytes and alexins are enabled to exert their protective powers. Resistance to poison must therefore be regarded as an important factor in general natural power of resistance.

II. SPECIFIC IMMUNITY

As a result of recovery from a specific disease or of protective inoculation.

A. SPECIFIC IMMUNITY *as a result of recovery from specific disease.*—Specific protection, lasting for a comparatively long time, is conferred after recovery from an acute eruptive fever such as small-pox, scarlatina, or measles, and also after typhoid fever; and in animals after recovery from cattle plague, contagious pleuro-pneumonia of cattle, sheep-pox, contagious pleuro-pneumonia of the horse, and black-quarter (G. *Rauschbrand;* F. *charbon symptomatique*).

There are other infective processes, however, which confer no protection, for instance, diphtheria, relapsing fever, and gonorrhœa, and after certain diseases, such as pneumonia and erysipelas, the individual appears to be even more liable to a subsequent attack. Malaria, too, confers no real immunity, yet the risk of acquiring pernicious rapidly fatal malaria is diminished after residence for a considerable time in malarious districts. Asiatic cholera is as yet believed to confer protection of only short duration. In the case of those specific infective processes which generally confer immunity, a slight attack affords as good a protection as would a severe one. This is probably the reason why, in the case of endemic infective diseases, such as typhoid fever or yellow fever, the inhabitants are usually less endangered than strangers, the former having probably previously suffered from the infection in a mild form.

In those instances where the blood serum of human patients who have recovered from cholera or typhoid fever has been examined, the same protective substances have been hitherto found as in animals which have been experimentally infected with cholera vibrios or typhoid bacilli. It is therefore advisable to deal under one heading with the conditions found in specific immunity in spite of their different modes of origin, for the specific immunity is essentially the same in both cases. It is always due to an *absolutely specific* protection against a certain infective agent or its poison, and is not due to a general increase of resistance. Not only so, but recovery from a specific disease may be followed by increased susceptibility to other infective diseases; thus a severe attack of measles is not infrequently the starting-point for tuberculosis.

B. PROTECTIVE INOCULATION (active or isopathic immunisation).—Since the discovery of antitoxins and antitoxic sera by Behring, two methods of artificial specific immunisation have been recognised: (1) *Protective inoculation,* whereby the specific protective substances have to be formed in the body itself; (2) Immunisation by transference of protective substances previously formed in *antitoxic serum.* Ehrlich designated the former method as *active,* the latter as *passive* immunisation. Behring has, however, of late preferred to term the former *isopathic* and the latter *antitoxic* immunisation. The chief difference between the two is as follows:

In isopathic immunisation, true protective inoculation, the individual must undergo a reaction, often a feverish one, after the introduction of the infective material. The antitoxin is meanwhile being formed, and protection is therefore obtained only after a period of time. The resulting specific immunity will, however, persist for a long time, usually for months, because the toxins which have been formed become bound to the tissue elements, and even after repeated severe venesections are not entirely got rid of in the withdrawn blood. In contradistinction hereto antitoxic immunisation by the introduction of serum induces specific protection at once without the production of any morbid symptoms; but the immunity conferred is of very brief duration (8 to 14 days), especially if blood serum of another species of animal be employed, for, being a foreign substance, it will be rapidly excreted. Such immunity, however, is of longer duration after the use of serum of the same species—in horses, for example, on using tetanus serum obtained from horses.

Just as in natural resistance we have to distinguish between resistance to bacteria and resistance to poison, must we do the same when dealing with specific immunity, and must therefore differentiate between "specific immunity to bacteria" and "specific immunity to toxin." Immunity to diphtheria is exclusively of the latter kind; diphtheria antitoxic serum has no action upon living diphtheria bacilli, it merely neutralises their poison. Diphtheria bacilli can even be cultivated in the specific curative serum. Diphtheria bacilli in a patient's body have to be overcome through the agency of the natural power of resistance, and this is easily effected if the toxins are rendered impotent by injection of antitoxin. The same holds good for tetanus. Behring, nevertheless, has recently advocated that there should also be local treatment of diphtheritic membranes, and especially those of the nose, with curative serum; and likewise in tetanus that the tetanus serum, or at least a portion thereof, should be injected as near as possible to the seat of infection if it be known.

If there be specific immunity to bacteria on the other hand, such as is induced by treating animals with cholera or typhoid bacteria, the serum of the immunised animal is merely bactericidal to the bacterium in question, and has no action on its integral cell poison. Hence if an animal previously treated with cholera vibrios be afterwards infected with a large number of the same, the latter will indeed perish, but a fatal intoxication may be induced by their cell poisons. This is one of the chief reasons of the failure to obtain an efficacious serum suitable for the treatment of cholera and typhoid in the human subject. Another point of importance is that in specific immunity to poison the condition is often a regional one, analogous to the condition dealt with when speaking of natural resistance to poison. Rabbits which, by means of long-continued treatment with bouillon containing the poison of tetanus, have become thoroughly immune to subcutaneous inoculation of tetanus toxin, are killed by intra-cerebral inoculation (Roux and Borrel). The conditions are similar in many other instances; for example, guinea-pigs in which a high degree of specific immunity has been induced by treatment with cholera vibrios are nevertheless not protected against intestinal infection with virulent cholera vibrios.

The following methods of active or isopathic inoculation may be differentiated:—

(1) Protective inoculation with living virulent virus.
(2) With living yet attenuated virus.
(3) With dead virus.

(4) With the bacterial cellular substances of the same.

(5) With dissolved toxic products of bacteria.

1. *Protective Inoculation with Living Virulent Virus.*—Inoculation for small-pox, which consists in the transference to healthy individuals of the contents of the variola pustule, was introduced into England in 1871 by Lady Mary Wortley Montagu, wife of the English ambassador at Constantinople, and was much used in England, Germany, and other countries in spite of its inseparable danger. Inoculation often caused severe or even fatal small-pox, and the disease was thus spread. The inoculated disease was, however, usually of a mild form, probably because inoculation into the skin affords conditions less favourable for the development of the small-pox virus than does natural contagion, in which instance the agent is probably carried from the mucous membranes into the blood and then multiplies there.

There has been much said of late regarding protective inoculation by means of subcutaneous injection of bouillon cultures of living virulent cholera vibrios, such as had been tried on a large scale in Spain by Ferran in the eighties. Cholera vibrios cannot multiply in the subcutaneous tissue; they die, are dissolved, and absorbed. In Ferran's inoculations the methods were imperfect and his statistics are quite unreliable. Haffkine, working much more accurately, has lately employed this method extensively in India, yet he generally uses dead cultures, and the subject is therefore discussed on p. 174.

In contagious pleuro-pneumonia of cattle, protective inoculation with the living virus (which has not yet been isolated) has been proved to confer effective protection, and is employed practically. Inoculation is performed at the end of the tail by subcutaneous injection either of lymph or fluid from the pulmonary tissue of a newly-killed animal suffering from the disease ("warm lymph"). The first result is swelling and inflammation resembling erysipelas around the seat of inoculation. The change often affects the whole tail, or extends even farther. There is in addition not infrequently necrosis and loss of the end of the tail, and also some feverish reaction. Animals thus treated prove immune to artificial and to natural infection of pleuro-pneumonia.

2. *Protective Inoculation with Living yet Attenuated Virus.*—In 1878 Buchner gave proof of the inconstancy of the anthrax bacillus when cultivated under definite conditions outside the body, but this inconstancy was not at that time made use of to confer protection. It is to the genius of Pasteur that we owe our knowledge of systematic protective inoculation by means of artificially induced attenuation. Pasteur's first success was gained in the disease termed fowl cholera (avian septicæmia); old cultures of fowl cholera bacilli, which have been exposed to the air and thus attenuated, were injected into the pectoral muscles of fowls, and induced a process of local inflammation and necrosis, which was followed by general specific immunisation of the birds.

The subsequent means of attenuation were the following:—

(*a*) *Attenuation by high temperature.*—Toussaint in 1880 demonstrated that anthrax blood loses its infective power by being heated to 55° C. for ten minutes; and in the following year Pasteur published his method of protective inoculation against anthrax, which is also founded on the power possessed by a high temperature (42°-43° C.) of attenuating anthrax bacilli. At this temperature anthrax bacilli can multiply, but cannot sporulate. Inoculation is commenced with the weaker cultures, which have been kept for twenty-four days at 42°-43° C. (first vaccine), and ten to fourteen days later a second and final injection is given, using the more powerful cultures which have been kept for twelve days at the above temperature (second vaccine). Animals thus treated are immunised against virulent anthrax. This method was stoutly opposed by R. Koch, yet was soon extensively employed in France and Russia, and afterwards in other countries, and favourable results were obtained in cattle and still more so in sheep. In Hungary, for example, according to Hutyra, 16,082 horses, 210,750 head of cattle, and 1,118,443 sheep were inoculated against anthrax from 1889 to 1893. The total loss in the first three years amounted to 2·18-2·44 per cent, and was only 0·61-0·55 per cent in the last two years. Opinions differ, however, as to the efficacy of this method, for the constancy of the vaccine is sometimes variable, and inoculation may therefore either confer no protection or may induce the disease and cause loss of stock. Again, the protection conferred even in the most favourable case is only of about one year's duration. Hence these protective inoculations should only be used in districts where anthrax is endemic and causes serious loss.

Arloing discovered a mode of protecting against *black-quarter* (G. *Rauschbrand*) which has proved of practical value. The dried and pulverised muscle-substance of affected animals is attenuated by heating to 100° C., is then mixed with sterile water, and injected into young cattle. It is usual to employ a weak infective material twice, and then a more powerful one. Favourable results were obtained in Germany (in Baden more particularly), Austria, and Switzerland. Kitt first modified this method by attenuating with steam under ordinary pressure, and afterwards by the use of pure cultures of Rauschbrand bacilli, which, on being subcutaneously inoculated into sheep and cattle, do not induce the disease, yet do confer immunity.

According to Kitt's statistics, the loss of stock from black-quarter was as follows:—

	Uninoculated.	Inoculated.
1886	107·5 per 1000	5·6 per 1000
1887	43·4 ,,	9·6 ,,
1888	78·8 ,,	11·1 ,,
1889	30·6 ,,	17·2 ,,
1890	33·8 ,,	2·5 ,,

In the year 1890, out of 1167 cattle inoculated once with Kitt's vaccine there was only one death, and this was attributed to the inoculation, whilst of 2803 head of cattle which had not been inoculated 44 died.

(*b*) *Passage through less susceptible Animal Species.*—An important step was gained when Pasteur achieved attenuation by means of *passage* through less susceptible animal species, as was first done in swine erysipelas. The principle was essentially the same as that of Jenner's vaccination, but this fact was not so clearly understood then as it is now. In 1789 Edward Jenner published the fact, which had long been popularly known in Gloucestershire, but which he thoroughly investigated in his own native place, that recovery from cow-pox affords protection against small-pox, and he also showed that cow-pox may be artificially transferred from one individual to another without losing its protective power. But there is now abundant proof that cow-pox is nothing but the variola of man attenuated by transference to the ox or calf. Jenner's vaccination soon came into general use, but it was then found that the protection conferred only lasts about ten to twelve years. The protective power of vaccination was proved by Jenner himself and his contemporaries by thousands of experiments with subsequent inoculation of small-pox. Another most convincing proof in favour of vaccination is found in the trivial mortality from small-pox in those countries in which, as compared with others, vaccination has been enforced by law. The experience gained in the Franco-Prussian war of 1871 was almost equivalent to that of an experiment on a large scale, for the German, in contradistinction to the French troops, had been vaccinated and for the most part revaccinated. The latter, during the campaign, lost 23,000 men from small-pox, whilst the Germans only lost a few hundreds, although their whole army was in France, where an epidemic of small-pox was raging among the civil population.

Now Pasteur, in the case of swine erysipelas, showed that the bacilli taken from the pig gain in virulence on transference to pigeons, but that they become less virulent after repeated *passage* through rabbits, and that they are then suitable for protective inoculation of swine. The last *passages* yielded vaccine No. 1, then twelve days later followed inoculation with the bacilli of pigeon erysipelas (vaccine No. 2). The animals are then highly immune, and *Der land- u. forstwirtschaftliche Kongress* at Vienna in 1890 warmly advocated the use of protective inoculation. It has been tried, more especially in Hungary, and the disadvantages of the method have since then become evident. Deaths from the disease are common among the more susceptible breeds of swine, and young swine which have been inoculated appear unsuitable for fattening. There is also a danger of swine erysipelas spreading because of the inoculations. Preference has, therefore, since then been given to other methods (*vide* p. 196).

(*c*) *Attenuation by Drying the Vaccine.*—The virus of rabies is not yet known, yet Pasteur's experiments, brilliantly conceived and patiently carried through, furnished us with means of prophylaxis and cure, which have long been recognised as of the greatest benefit to mankind. Pasteur first found that the virulence of rabic virus is exalted by successive subdural *passages* through rabbits, whilst weakened by *passage* through apes. He strove to make use of this latter fact in his attempts to immunise dogs, but as the results obtained were so inconstant he forsook this apparently hopeful line of research for a new method of attenuation, that by drying.

By drying in an atmosphere freed from water and carbonic acid he succeeded in attenuating the highly virulent spinal cords of rabid rabbits, in which the incubation period has been shortened from about fourteen to seven days by numerous *passages* ("fixed virus"), and he was able to attenuate the cords to any degree proportionate to the duration of the desiccation, or even to render them completely inactive, and thus to transform them into a vaccine. Animals which were at first inoculated with very weak virus could withstand in succession the stronger, and lastly the most virulent spinal cords without becoming diseased. Pasteur then employed this method for the treatment of persons bitten by rabid dogs, wolves, etc., for he rightly supposed that a protective inoculation may be possible and successful even subsequent to infection, if the infective process be at first a localised one and pursue a chronic course. The Pasteur treatment of rabies is therefore not a true curative method, but essentially an active immunisation. To attenuate the spinal cord by desiccation it is kept at 22° C. in a vessel, the bottom of which is covered with fragments of caustic potash. A cord kept here for one to four days still causes rabies within seven days; by the use of a cord dried for more than five days the incubation period is prolonged, and a cord dried for twelve to fourteen days has become inactive. The treatment of the human subject commences with the injection of 1-3 cc. of an emulsion of completely attenuated cord, which is prepared by the trituration of 1 c.m. of cord in 5 cc. of indifferent fluid; and within twenty days gradually increasing the strength of the dose till a cord dried for two days is used. Complete immunity is only attained three to four weeks after commencement of the treatment, hence it is important to begin the latter as soon as possible. Deaths from rabies after the treatment has been carried out cannot consequently be included in any statistics unless the treatment has been completed at least fourteen days previously, for complete protection can only then be hoped for. The treatment has been gradually rendered more simple and efficacious, for instead of injections being often repeated and the degree of virulence slowly raised, fewer injections and a more rapid increase of virulence are now employed. The results in consequence are gradually becoming more favourable, and between 1886 and 1890 the returns from the Pasteur Institute in Paris are as follows :—

1886,	treated	2617 ;	died,	25 = 0·94	per cent.
1887	„	1770	„	13 = 0·72	„
1888	„	1622	„	9 = 0·55	„
1889	„	1830	„	6 = 0·33	„
1890	„	1540	„	5 = 0·32	„

As regards the locality of the bite, on which depends the danger of infection, the statistics of the same Institute for 1890 are as follows :—

Bite on head,	mortality,	0·85	per cent.
„ hand,	„	0·45	„
„ limbs and trunk,	„	0·	„

Institutes of preventive medicine formed after the model of the Pasteur Institute in Paris have been gradually founded in every country, and recently also in Germany, where in consequence of police regulations rabies is comparatively rare. The results are everywhere very favourable, and the average mortality is less than 0·2 per cent.

(*d*) *Attenuation by the Addition of Antiseptics to the Nutrient Medium.*—Virulence can be reduced by the cultivation of infective agents on media to which a weak antiseptic has been added. The sporulation of anthrax bacilli can be permanently inhibited by growth in carbolised bouillon, hence the bacilli can be attenuated to any extent. Such methods possess as yet no practical interest.

3. *Protective Inoculation with Killed Cultures.*—In many cases the same degree of immunity may be conferred by the use of dead cultures as by the use of living cultures of the virus, and the method is safer and simpler. Such is the case in cholera, typhoid fever, and plague.

(*a*) CHOLERA.—Protective inoculation against cholera originated in R. Pfeiffer's discoveries regarding the immunisation of guinea-pigs by treatment with cholera cultures. Living cholera vibrios are injected into the peritoneum of such an immunised animal, and drops of the exudation are from time to time withdrawn by glass capillary tubes and examined in either a fresh or stained condition. The vibrios are seen to lose their motility almost immediately, after ten minutes they swell up, and ten minutes later break up into little balls resembling micrococci, which after another twenty minutes can no longer be seen, and have apparently been completely dissolved in the peritoneal exudation. The same process is seen if the serum of the immunised animal or the blood serum of a patient convalescing from cholera be mixed with living cholera vibrios and injected into the peritoneum of a normal guinea-pig. The normal animal now acts like an immunised one, the cholera vibrios soon die, and the animal remains alive whilst control animals succumb (Pfeiffer's reaction). As the destruction of the vibrios is a rapid one only when the animal has been treated with *true* cholera vibrios, and not merely with those which are similar, yet specifically different, this reaction can *vice versa* be employed for the diagnosis of true cholera vibrios provided we have a supply of true cholera immune serum.

Guinea-pigs may be immunised not only by living cholera vibrios, but also by the use of cultures which have been killed either by heating to 56° C. for one hour, or by chloroform vapour. In the protective inoculation of man the employment of dead cultures, to which 0·5 per cent of carbolic acid may be added without impairing their activity, is obviously of great advantage.

The substances which are called into action in the serum of the immunised animal body were termed "*bacteriolytic*" or "*specific bactericidal*" substances by R. Pfeiffer, to distinguish them from the bactericidal alexins of normal serum, for the action of the former is strictly specific, *i.e.* directed only against cholera vibrios. This immune serum has no antitoxic action whatever on the poisons contained in the cholera vibrios. The action is merely an antibacterial one leading to destruction of living cholera vibrios, whilst it is always possible, by large quantities of a virulent killed cholera culture, to *poison* an animal immune to cholera, as already mentioned.

Cholera immune serum can act even in extreme dilution. The degree of potency is tested by mixing a given quantity of serum with five to ten times the minimum lethal dose of an agar culture of virulent cholera vibrios, and injecting the mixture into the peritoneum of guinea-pigs. Pfeiffer's standard is the smallest quantity of serum which in one hour and within the peritoneal cavity will dissolve that quantity of vibrios, the weight of the guinea-pig employed averaging 250 grammes. It is now definitely ascertained that as regards the bacteriolytic substances of the immune serum we have, as a matter of fact, to deal with two different substances, the joint action of which leads to the dissolution of cholera vibrios. Such a result may be seen not only in the peritoneum of guinea-pigs, but also outside the body in reagent glass or hanging drop, if we employ absolutely fresh immune serum, or, to one no longer so, add a drop of fresh normal serum (Bordet). Pfeiffer showed that even on the introduction of old and hence apparently inactive serum, or of immune serum which had been heated to 60°-65° C., into the peritoneum of a normal animal, vibrios are dissolved there. This was formerly regarded as due to a sort of "regeneration" of the true specific anti-substance which is preserved in spite of heating to 65° C., but we now know that the phenomenon is due to the presence of the normal alexins and to a combined action. By the action of the specific anti-substances, retained after heating to 65° C., the cholera vibrios become predisposed to the solvent action of the alexins. The French, therefore, often designate this anti-substance as "substance sensibilitrice."

The formation of these specific anti-substances in the animal body has hitherto been shown to occur chiefly in the spleen, bone-marrow, and lymphatic glands (Pfeiffer, Marx, Wassermann).

After these discoveries had proved the possibility of conferring to men and animals definite protection against cholera, Haffkine in India carried out similar protective inoculations with cholera vibrios in more than 40,000 persons.

Emulsions of agar cultures of living cholera vibrios are injected subcutaneously. His first vaccine is $\frac{1}{12}$th of an agar culture of dead vibrios, or of vibrios which have been attenuated by cultivating a series of agar subcultures at 39° C. The second vaccine, $\frac{1}{12}$th of an agar culture of vibrios rendered virulent by *passage*, is afterwards injected. A few hours after the injections the temperature usually rises 1°-2° C., but returns to normal in twenty-four hours. Such a reaction with local pain and swelling, with rigor, depression, and anorexia, is, however, not an effect peculiar to cholera vibrios, but follows upon the subcutaneous injection of all bacterial emulsions, and even of innocuous and killed bacteria (Buchner, 1890). Kolle then demonstrated that one injection of a dead cholera culture—2 mg. fresh agar culture suspended in 1 cc. of bouillon—causes the same effect as repeated inoculations, and that the bacteriolytic power of the serum beginning to rise after the fifth day reaches its height on the twentieth. Protection due to preventive inoculation may therefore be looked for after the fifth day, an expectation which harmonises with actual results so far.

The results of preventive inoculations are on the whole favourable, even though, owing to the existing difficulties, exact statistical proof of the total morbidity and mortality of inoculated and non-inoculated be not forthcoming. Yet there are many observations indicating its value. In Calcutta in 1894, a record was made of every case of cholera occurring in thirty-six houses with a total of 521 inhabitants: 181 had been inoculated before the outbreak of the epidemic, some a considerable time previously, and of these 4 (2·2 per cent) were attacked and died of cholera; 340 had not been inoculated, and 39 of them (11·6 per cent) died. In one of these houses there were 18 inhabitants; of the 7 uninoculated persons 4 were attacked and 3 died, and not one of the 11 inoculated individuals was attacked. During the cholera epidemic of "The Gya Jail" inoculation was only commenced after the beginning of the outbreak. In 287 uninoculated persons the final mortality was 4·9 per cent, being 2·4 per cent among the inoculated. We must assume that these protective inoculations, to a certain if not a great extent, do not act by an accumulation of specific anti-cholera substances in the body, but by an increase of natural general resistance. That this assumption is correct is also indicated by Haffkine's observations, namely, that protection does not last long, and has at any rate completely vanished after fifteen months.

A very effective anti-cholera serum can be obtained from animals—guinea-pigs, rabbits, and goats—by treatment with dead cholera vibrios. It is indeed of no value in the treatment of cholera, but is so for the differential diagnosis of cholera vibrios from other closely related forms. Goats yield a particularly potent serum, but rabbits are most suitable, for even on the fifth day after a single injection of about three agar cultures, previously killed at 60° C., they yield a serum which even in solution of 1-200 is suitable for diagnostic purposes. The diagnosis of cholera vibrios by means of such a cholera immune serum is made either by observing Pfeiffer's phenomenon, *i.e.* the rapid dissolution of vibrios introduced with immune serum into the peritoneum of a healthy animal, or more simply by the specific agglutination discovered by Gruber and Durham.

The phenomenon of *agglutination* consists in the fact that cholera vibrios brought into contact with cholera immune serum in a test-tube lose their active motility, and, adhering to one another, collect into little clumps. The clumps soon become larger, and form first small and then larger flakes, which are deposited as a sort of sediment at the bottom of the fluid, which now becomes clear. Other observers had previously seen similar phenomena, but had not recognised their diagnostic significance. Gruber and Durham were the first to describe agglutination accurately, and to regard it as a fundamental action of specific bacteriolytic immune serum, and their statements were immediately afterwards confirmed and amplified by the writings of R. Pfeiffer and Colle, and Pfeiffer and Vagedes.

Agglutination is not only seen with cholera vibrios. If animals have been treated with certain forms of vibrios, typhoid bacilli, B. coli or B. pyocyaneus, their blood-serum even in very small quantities will, according to Gruber and Durham, respectively agglutinate these different bacteria in suspension. The phenomenon can either be observed macroscopically in tubes or microscopically in hanging drops, the latter being the finer method of the two. In Gruber's reaction, in short, the different bacteria are thrown down by their respective specific immune serum. Yet, according to Gruber, the reaction is not an absolutely specific one, since powerful cholera immune serum, for example, can also agglutinate other allied forms of vibrios. Yet an immune serum always acts most effectively (*i.e.* is still potent when greatly diluted) on its respective specific bacterium. It is still doubtful whether the agglutinins—those substances of the immune serum which cause surface adhesion of bacteria—are identical with the true specific anti-substances of the immune serum, or whether both substances exist independently in the serum. The agglutinins, at any rate, as compared with the alexins, exhibit the same resistance to a temperature of 60° to 65° C., to putrefaction and to the influence of light as the true specific anti-substances, and therefore closely resemble the latter. Cases, however, have been observed where the serum possessed distinct power of agglutination, yet no specific protective power.

A point of importance is that the agglutinins disappear, or are used up during the reaction (Gruber), just as occurs if bacteria are cultivated in their specific agglutinating serum (Pfeiffer). In both cases the agglutinins are directly absorbed by the specific bacteria, as has been lately proved by the fact that the absorbed agglutinins can be redissolved from the bacteria by dilute alkalies, and separated by centrifuging (M. Hahn and Trommsdorff). The agglutinin solution thus obtained can again agglutinate specific bacteria added to it.

(*b*) TYPHOID.—The immunisation of animals against typhoid bacilli is performed in the same way as against cholera vibrios (R. Pfeiffer). Agar cultures of typhoid bacilli, one day old, are suspended in bouillon, are killed by heating at 65° for one hour, and are once or repeatedly injected. The serum of the animal thus treated then exhibits a specific bactericidal action on typhoid bacilli, *i.e.* if living typhoid bacilli are injected together with this immune serum into the peritoneum of a normal guinea-pig they are rapidly destroyed, whilst without the immune serum the same bacteria would multiply and cause the death of the animal. Such an experiment is still successful if the specific immune serum has been heated to 60°-65°. The true active anti-substance is therefore preserved in the typhoid immune serum, even at the above-mentioned temperature, and the normal animal body has only to supply the requisite alexin, whilst the specific anti-substance predisposes the bacteria to the influence of the latter. The conditions are therefore analogous to those of cholera.

In the human subject a certain degree of protection can be conferred against typhoid as against cholera, and the serum then gains distinct specifically bactericidal power. A single injection of 2 mg. of a dead agar culture ($\frac{1}{10}$th of a culture), to which 0·5 per cent of carbolic acid has been added, is sufficient for an adult. Injection under the skin of the back is followed by transient elevation of temperature to 38·5° C., headache, and depression; the seat of injection and the neighbouring lymphatic glands are for a few days very tender to pressure. The blood, even a week afterwards, may act specifically on typhoid bacilli, for when such serum even in a dilution of 1-200 is mixed with typhoid bacilli and injected into the peritoneum of guinea-pigs the bacilli are quickly destroyed. Such serum, however, does not always agglutinate *in vitro.* It is not yet known whether such inoculation affords the human subject protection from typhoid when infection has occurred in the usual manner *per os.* Soldiers on their arrival in India have of late been inoculated for typhoid by Haffkine, but the results are not as yet decisive.

Treatment of animals, guinea-pigs, rabbits, and goats does not furnish a serum as active against typhoid bacilli as was the case in cholera. Yet rabbits after a single injection of three killed agar cultures usually furnish a serum which agglutinates in a dilution of 1-150. The power of agglutinating typhoid bacilli possessed by the serum of animals treated with typhoid bacilli, or of persons who have recovered from typhoid, is of great importance for the differential diagnosis of these bacilli. The phenomenon is the same as in the case of cholera (Gruber and Durham). The reaction is, however, not *absolutely* specific, for typhoid immune serum agglutinates not only true typhoid bacilli, but also B. coli and B. enteritidis Gaertneri. But whilst true typhoid bacilli are agglutinated by a powerful typhoid immune serum, even in a dilution of 1-100 or 1-500, a more concentrated serum —a dilution not greater than 1-30 or 1-50—is always necessary to agglutinate these other bacteria. The phenomenon of agglutination acquired great practical importance from the clinical standpoint by Widal's discovery that typhoid bacilli are agglutinated not only by the serum of persons who have recovered from typhoid, but also as a rule by that of typhoid patients in the first few weeks or days of their illness. This method of diagnosis ought to be termed the Gruber-Widal reaction, for it is merely the clinical application of the phenomenon of agglutination first discovered by Gruber.

The Technique of the Gruber-Widal Reaction.—The serum of the suspected typhoid patient is mixed with typhoid bacilli, which have been grown on agar and are suspended in bouillon. The result is watched at 37° C. either in a tube or hanging drop. The blood used is obtained by puncturing the tip of the finger or lobe of the ear, is drawn into a sterile pipette, and then blown into a small narrow sterilised tube, which is laid horizontally till coagulation begins and then placed upright. In a few hours sufficient serum will have collected at the bottom of the tube, or the separation may be hastened by centrifuging. The serum should then be at once diluted with 50 parts of bouillon, for normal serum in a concentration greater than this will sometimes agglutinate. The serum of typhoid patients will usually agglutinate when greatly diluted (up to 1-5000), but a marked agglutination with 1-50 is sufficient for diagnostic proof, provided that it occurs at about 20° C., within one or at most two hours. The typhoid culture employed must not be more than twenty-four hours old and of moderate virulence. The reaction is characterised by the formation of first smaller and then larger clumps, whilst a control test with the same typhoid emulsion does not show this phenomenon in the same period of time.

The serum of typhoid patients sometimes gives this reaction on the second or third day, but usually only later and sometimes only during convalescence. The agglutinative power may, however, persist for a long time after recovery from typhoid, even though the attack be mild, and this may give rise to errors in diagnosis. That there is really a fresh attack of typhoid may be proved by observing on repeated quantitative determination that the agglutinative power is increasing. The severity of an attack cannot be deduced from the intensity of the agglutination. At the height of the disease the agglutinative power may sometimes be reduced; and in a mild attack the serum may sometimes agglutinate very markedly. The negative result of one examination does not exclude the possibility of typhoid; examination should be again undertaken, as it is often only in the later stages that the serum has the power of agglutinating.

The agglutinative power of a typhoid serum is usually but not always proportionate to its specific bactericidal action on typhoid bacilli. Thus the serum of dogs after the introduction of typhoid bacilli by the mouth may agglutinate and yet have no specific bactericidal power, which it does, however, possess if the typhoid bacilli have been inoculated into the peritoneum.

(*c*) PLAGUE. — By the repeated intra-venous or intra-peritoneal injection of plague cultures killed at 58° a serum can be obtained from rabbits. Small doses of this serum protect other rabbits against virulent plague bacilli, even though used twelve hours subsequent to infection (Yersin, Calmette, and Borrel). Yersin based his method for the production of plague serum on this fact (*vide* p. 193).

In view of the fact that natural recovery from plague confers a definite, though only a relative, immunity to plague, Haffkine in 1897 tried direct active immunisation of man by means of killed plague cultures. The method consists in the subcutaneous injection of bouillon cultures of plague bacilli, one month old, killed at 70°. For an adult an injection of 2-3 cc. is made into the upper arm or abdomen, and is followed by swelling and tenderness at the seat of injection and by slight fever. This injection ought to be followed by a stronger dose after eight or ten days, but few people can be induced to submit to a second inoculation. During the plague epidemic in India Haffkine's inoculations have been performed on a large scale and with favourable results. When plague broke out in the prison of Byculla in Bombay, and after fifteen cases, eight of them fatal, had occurred between 23rd and 30th of January, Haffkine on the evening of the 30th of January inoculated 154 prisoners, who subjected themselves voluntarily, giving each one 3 cc. Up till the 6th of February there were only two cases of plague, neither being fatal, among these 154 persons situated in the same circumstances and living together with 177 non-inoculated. Of the latter, 14 were attacked and 6 died. During the plague epidemic of 1897 in the Portuguese town of Damaon, it is recorded that among 62 families the mortality from plague was 8 per cent in 250 inoculated individuals, and 29·8 per cent in 124 non-inoculated. In inoculated persons, moreover, the course of the disease is said to be milder and the buboes suppurate more rapidly. Further statistics have also indicated the remarkable reduction of mortality among the inoculated, and active immunisation with killed plague bacilli must therefore be strongly recommended, more especially for the protection of physicians, nurses, persons who have to carry out disinfection, and so on.

Immunity by this method is only obtained after about a week, as has been definitely proved by animal experiment, but it then lasts for months. According to the researches of the German Plague Commission, the immunising substances are contained in the cell substances of the plague bacilli, and not merely in the filtrates. The use of bouillon cultures is therefore of no special value, and it is better to use agar cultures which have been suspended in physiological saline solution, and then killed by being heated for one hour at 65° C. Heating to the boiling-point would destroy the immunising substances of the plague bacilli in contradistinction to those of typhoid and cholera bacteria. Before using this emulsion as a vaccine it is advisable to add 0·5 per cent of carbolic acid, which does not impair the power of the immunising substances. Larger quantities of plague bacilli can be injected into man than typhoid bacilli, for the former are less poisonous.

Plague immune serum obtained by the treatment of animals with killed cultures has the power of agglutinating, and as a rule, but not invariably, that of immunising.

4. *Protective Inoculation with Bacterial Cell Substances.*—In different instances, for example, cholera, typhoid, and plague, we have seen that active immunisation may be effected by the killed bacterial cells, whilst the associated filtrates are inactive. In these instances, therefore, the substances conferring immunity must be contained in the bacterial cells themselves. The bacterial cell substances, which are mainly of a proteid nature, are obtained—(*a*) By extraction with boiling water, whereby albumoses—so-called bacterial proteins—are formed (the older tuberculin, mallein); (*b*) or without induction of chemical change by purely mechanical pulverisation of the bacterial cells (tuberculin TR, bacterial plasmin).

(*a*) *Immunisation with Bacterial Proteins—Tuberculin.*—Koch's tuberculin is obtained by concentrating at the boiling-point cultures of tubercle bacilli in glycerine bouillon. The proteids of the tubercle bacillus are in consequence partly dissolved, and they are purified by repeated precipitation with 60 per cent alcohol. The active principle, therefore, consists in the dissolved proteins of the tubercle bacillus which resemble albumose. Subcutaneously their action is similar to that of other bacterial proteins, causing hyperleucocytosis which in man is merely local, swelling, redness, tenderness, and with stronger doses fever, as previously mentioned. But as even a very minute quantity of tuberculin is sufficient to cause a reaction in the body, we assume that it has a specific action, as well as the general action of a bacterial protein manifested by increase of natural resistance to bacteria. Tuberculin has much less effect on healthy than on tuberculous guinea-pigs. The former can stand a subcutaneous dose of 1-2 cc., whilst the latter invariably succumb to 0·5 cc. Man is much more susceptible; in a healthy adult even 0·25 cc. causes great rise of temperature, severe local pain, general depression, and so on, whilst after 0·01 cc. the symptoms are as a rule only trivial. This dose given to a tubercular patient, however, usually causes severe fever, lasting twelve to eighteen hours, and at the same time a well-marked local reaction in tubercular areas, which is specially evident in tuberculosis of the skin, lupus, but which occurs in tuberculosis of the internal organs too. A few hours after the administration of tuberculin the tubercular areas commence to swell, and there is marked increase of blood-supply within them and in their immediate vicinity.

In consequence of the great difference in the susceptibility of tubercular and of healthy people to tuberculin, its use is of considerable importance for the diagnosis of latent tuberculosis. The normal temperature having previously been accurately ascertained, an adult receives a first injection of 1 mg. tuberculin, a few days afterwards 5 mg., and still later 10 mg. The corresponding doses for children under 10 years of age are 0·5, 1·0, and 5·0 mg. The temperature must be taken every three hours, and an elevation of 0·5° C. above the normal temperature of the individual indicates a positive reaction. This diagnostic procedure ought to be employed more than it has hitherto been. If the reaction be doubtful the same dose is to be repeated, or a larger one may be given. No injection should ever be given to a feverish patient. Tuberculin remains unchanged for a long time because it contains a large amount of glycerine.

In veterinary medicine tuberculin has for years been in general use as a diagnostic agent, and especially for the diagnosis of tuberculosis in cattle. The dose for a cow is 0·5 cc., and it is only a greater rise of temperature than 1° C. which can be regarded as positive proof of tuberculosis. There will be few errors in diagnosis if the diagnostic inoculations and the post-mortem examinations of the animals are accurately and carefully made.

Out of 124 animals which had previously reacted to tuberculin Nocard found 123 to be tuberculous, although the disease was often localised and obscure. These diagnostic inoculations have been much used on a large scale in Denmark and France, and have been an excellent means of weeding out tuberculous animals.

R. Koch recommended tuberculin for therapeutic as well as diagnostic purposes; at first it was taken up enthusiastically, but it was afterwards as severely discarded, yet the condemnation of tuberculin has probably been too pronounced. If guinea-pigs infected with tuberculosis are given an injection of dilute tuberculin every day, or every second day, their condition is improved, and death is deferred from the ninth to the nineteenth week. After death animals thus treated show marked pulmonary tuberculosis, whilst the tuberculosis of the abdominal organs is undergoing cure. Control animals not treated with tuberculin soon die from tuberculosis of the liver and spleen; pulmonary tuberculosis, which is a late manifestation after inoculation with tubercular bacilli, has not had time to develop in them. This favourable effect of tuberculin is not due to a direct action on the tubercle bacillus, but to the reaction—combined with hyperæmia—of the tissues which are the seat of tuberculosis. We have already indicated that tubercular lesions in an organ are cured by a permanent increase of blood in the latter. Hence tuberculin is merely a means of inducing local hyperæmia of tubercular foci.

In spite of these experimental results most physicians have had an unfavourable experience of tuberculin as a curative agent. This is due to the fact that pulmonary tuberculosis in man, whenever it has been detected in consequence of fever, almost always represents a mixed infection with streptococci, and tuberculin treatment is unfavourable in the case of a streptococcal infection. But pure tuberculosis (simple tuberculosis) is similar to the artificially induced tuberculosis of guinea-pigs, and can be decidedly improved and not infrequently cured by tuberculin. Petruschky has lately reported many favourable cures. He recommends an intermittent method of treatment, *i.e.* regular alternation between two to three months' use of tuberculin and three to four months' pause. Alternation is necessary because tolerance to tuberculin is induced, and it therefore ceases to act. To effect a cure, it is said that treatment must on an average be continued for two years.

Mallein.—According to Nocard mallein is obtained by heating, by concentration to $\frac{1}{10}$th of its previous bulk, and by filtration of glycerine bouillon cultures of B. mallei one month old. Mallein is thus prepared in the same way as the older tuberculin, and it too belongs to the bacterial proteins. Opinions differ as to its value in diagnosis, for non-glandered animals sometimes react to mallein. Nocard considers that only those animals which yield a distinct reaction—a greater rise of temperature than 2°—in combination with definite clinical symptoms are to be regarded as affected with glanders, and therefore destroyed. The action of mallein, too, is not an absolutely specific one, for other bacterial proteins can cause similar although weaker effects.

(*b*) *Immunisation with Bacterial Cell Substances obtained mechanically.*—*Tuberculin TR.*—R. Koch's more recent tuberculin TR (1897) is obtained from virulent cultures of B. tuberculosis by drying in an exsiccator, by fine mechanical pulverisation and extraction with distilled water. The extract is divided, by centrifuging, into an upper fluid portion and a solid sediment. The latter, after being again dried, pulverised, and extracted with water, constitutes tuberculin TR in which glycerine induces precipitation, whereas the older tuberculin contains much glycerine.

According to Koch, tuberculin TR has the definite power of inducing specific immunity, which is said to be developed without any reaction. It is said that as soon as insusceptibility to tuberculin TR is gained there is also immunity to the older tuberculin and to the tubercle bacillus. Tuberculin TR, which, as well as the older tuberculin, is prepared at the manufactories at Höchst-a.-M., contains 10 mg. of solid substance in 1 cc., and for use is diluted with physiological saline solution. The first dose is usually 0·002 tuberculin, being equivalent to $\frac{1}{500}$th mg. solid substance. Injections are then given every second day in gradually increasing doses, care being taken to avoid any rise of temperature greater than 0·5° C. The dose is gradually increased till 20 mg. is given. With tuberculin TR, Koch was able to immunise animals, or to cure those previously infected. But tuberculin TR, as used in medical practice, has yielded results almost as unsatisfactory as those of the old tuberculin, and another disadvantage of the new preparation is that it can only be kept for a short time.

Bacterial Plasmin.—Mechanical pulverisation of the cells of the lower fungi, as a mode of obtaining their intra-cellular substances, had been employed by E. Buchner in the Hygienic Institute in Munich before Koch's publication on the tuberculin TR. It is this method which furnishes the main proof that the true fermentative substance, the zymase, is contained in a soluble form in the cell juice—plasmin—of beer-yeast cells. Pure yeast is mixed with infusorial earth and quartz-sand, thoroughly pulverised, and then exposed to hydraulic pressure of 300-500 atmospheres. The juice, which contains much albumin, is filtered and freed of all living cells, and it undergoes true alcoholic fermentation on sugar being added. We must assume that the zymase is a product of and is contained in the yeast cells, and that the fermentative energy of the yeast depends upon the production of zymase.

As the cell-juices or plasmins of the lower fungi can be obtained by this method in an unchanged and active form, M. Hahn, at the Hygienic Institute, Munich, therefore prepared the plasmins of cholera, typhoid, tubercle, and other bacteria, and employed them for immunisation. A single injection of 0·5 cc. of "cholera plasmin" appeared well adapted for immunising guinea-pigs against intra-peritoneal infection with ten times the fatal dose of living virulent cholera vibrios, and the immunity lasts at least three to four months and is specific. The action of "typhoid plasmin" is analogous. The sera of animals treated with either of these plasmins have a powerful specific agglutinative action. "Tuberculo-plasmin" after filtration is a clear pale yellow fluid containing nucleo-albumin, and it will keep for a considerable time after the addition of 20 per cent of glycerine and 5 per cent of common salt. This preparation has been used with favourable results in the treatment of guinea-pigs infected with tuberculosis, but in only a few cases has it been hitherto employed in the human subject.

5. *Immunisation with Toxic Filtrates of Bacterial Cultures.*—In 1886 Salmon and Smith showed that pigeons can be rendered immune to hog-cholera (hæmorrhagic septicæmia) by treatment with filtrates of hog-cholera microbes. Foà and Bonome, Beumer and Peiper, and lastly Charrin in 1887, performed similar experiments in the diseases of rabbits artificially induced by proteus and B. pyocyaneus, Roux and Chamberland in malignant œdema, Roux (1888) in black-quarter (*Charbon symptomatique*), C. Fränkel in diphtheria, and so on.

It is especially in the latter cases that we are concerned with the production of a well-marked specific tolerance of poison. The nature of this tolerance was only made evident after the fundamental facts regarding bacterial toxins had been made known by Roux and Yersin. They showed in 1886, as a result of splendid research, that the poison of the diphtheria bacillus possesses special properties, and more especially extreme susceptibility to heat—being destroyed at 65° C.—and again the capability of being carried down mechanically by chemical precipitates (calcium phosphate) in the filtered cultured fluid, properties which had until then been recognised mainly in the digestive enzymes. Brieger and C. Fränkel confirmed and amplified these facts, and showed that the poisons of diphtheria and tetanus can be obtained in a moderately pure form by means of a method as simple as possible, *i.e.* from filtered cell-free cultures by simple precipitation with absolute alcohol. These poisons gave the reactions of albuminous substances, and were therefore termed "tox-albumins." But as Brieger has since said that they are not albuminous, it will probably be best to term them *specific toxins*, in contradistinction to the non-specific toxins resembling ptomaines, *e.g.* cadaverin, putrescin, etc. Specific toxins are characterised by the fact that they can cause in an animal all the symptoms of the infection in question, *e.g.* tetanus.

In regard to the origin of toxalbumins, Brieger and C. Fränkel believed that they were split off from the proteids of the nutrient medium by the action of the bacteria. This is not so, for if diphtheria and tetanus bacilli be successfully cultivated in nutrient fluids containing no proteid, toxins are still formed (Guinochet, Buchner). Hence the specific toxins must originate directly from the bacterial cells; they are products of the specific plasma of the bacteria, and are excreted during the life of the latter.

Now the specific toxins are to be regarded as the really active principle in the production of specific immunity to poison, although we have to note that solutions containing the poison alone are not suitable when starting immunisation (Roux and Yersin), and again that fluid cultures of diphtheria and tetanus which have lost their poisonous character will confer immunity. These phenomena are accounted for by the presence of modified toxins—which Ehrlich calls "toxones"—which are not themselves poisonous, yet bring about the formation of antitoxins. When an animal is to be immunised, it is advisable, the true poison being too dangerous, to make a start with its non-poisonous modifications which are contained along with modified poisons in the cultures.

After the discovery of diphtheria and tetanus poisons the first attempts at immunisation were unsuccessful as the animals died of chronic poisoning. Tolerance to poison was only gained after the filtrates had been weakened by heating to 60° C. (C. Fränkel) or after the addition of certain chemicals (*e.g.* iodine terchloride, Behring), and the toxins thus destroyed whilst the toxones remained. When once the animals had gained a certain degree of tolerance of poison, poisonous filtrates could then be employed in order to render the tolerance greater and more powerful.

Apart from diphtheria and tetanus attempts were afterwards made to isolate a soluble specific cholera toxin from cultures of cholera vibrios, and thereby to immunise animals to this poison (Behring and Ransom). Specific toxins were similarly isolated from typhoid bacilli, pneumococcus, plague bacillus, etc., but they have hitherto been of little practical value.

Direct active immunisation to poison is not generally applicable for practical purposes. This is partly because of its associated difficulty and danger, and still more so because in the case of diphtheria and tetanus we must confer rapid protection, and immunisation by injection of filtrates is but a very gradual process.

The great significance of active immunisation to poison by the use of toxic filtrates of bacterial cultures depends entirely on the discovery, made by Behring and Kitasato in 1890, of the antitoxic value of the serum of an animal immunised to poison (*i.e.* to diphtheria and tetanus toxin). It appeared that the protection from poison obtained by specific treatment can be transferred by the serum to other animals, and that they too are then immune to this specific poison (*passive* immunisation).

Just as immunity to toxic bacterial products can be induced, so can immunity to vegetable toxalbumoses be obtained by the use of these latter substances. By feeding with ricin, abrin, and robin, Ehrlich induced, even in the most susceptible animals, a high degree of immunity to these poisons, and similarly marked immunity to snake venom may be induced in various animals (Calmette, Physalix, and Bertrand).

C. SPECIFIC IMMUNITY BY THE TRANSFERENCE OF BLOOD-SERUM OF ACTIVELY IMMUNISED ANIMALS—PASSIVE (ANTITOXIC) IMMUNISATION—SERUM THERAPEUTICS.—The most rapid means of conferring specific immunity, and especially specific immunity to poison, is based on Behring's discovery, in 1890, of the transference of blood-serum of specifically immunised animals to other animals or man. It was in diphtheria that Behring first proved that the blood and serum of animals immunised to diphtheria is capable of at once rendering other animals insusceptible to what would otherwise be a fatal dose of diphtheria poison, or, as he expressed it, "of destroying diphtheria poison."

In conjunction with Kitasato he afterwards proved the same for tetanus. The transference of immune serum in both cases first confers protection against the specific poison, and thus indirectly against infection with the living virus, for if their poisons are inactive both diphtheria and tetanus bacilli are overcome and destroyed by the natural power of resistance of the body. As the immunising anti-substances are contained in the immune serum they do not require to be formed in the new animal body; in passive immunisation, therefore, the new animal body does not require to undergo any reaction, for the effect is immediately induced. But another difference from active immunisation is the very short duration of the immunity conferred by transference of serum, for foreign serum does not remain long in the body, but is destroyed or excreted within eight to fourteen days. As compared with active immunity, therefore, passive immunity is only a transient one.

Passive immunisation has been performed not only in diphtheria and tetanus, but in a series of other infective processes, and has to some extent yielded results of practical value. Again, the active immunity to poison conferred by treatment with ricin, abrin, and robin can be transferred by the serum to other animals (Ehrlich). Ehrlich has also shown in these and other instances that it is not the serum, but the milk, which may be the vehicle for the transference of antitoxin, and that immunity may thus be transmitted from the mother to the offspring by suckling (*vide infra*). The transference of blood-serum does not only confer protection from subsequent infection, but, provided the conditions are otherwise favourable, it also induces a cure after precedent infection. It is experimentally proved that the resulting effect is essentially the same whether we inject a mixture of antitoxic serum and toxin, or inject each of them into different parts of the body, or lastly, whether we inject the serum twenty-four hours before the toxin. It is a very different matter, however, if the antitoxic serum be injected subsequent to infection. The greater the delay in the introduction of the serum the more unfavourable is the prognosis, the greater is the quantity of serum required, and the more uncertain is the result (*vide infra*).

1. *Mode of obtaining Antitoxic Serum.*—According to Behring the animals which are to furnish the serum (horses) are rapidly and safely immunised (*a*) by using unaltered bouillon cultures of tetanus bacilli—their filtrates are preferable—injecting $\frac{1}{20}$th of the minimum fatal dose at first and increasing to double this dose within four weeks; (*b*) or, as is better, by starting the treatment with larger quantities of cultures rendered less poisonous by the addition of suitable chemicals, such as iodine terchloride.

Behring gives the following special directions for the immunisation of horses: 200 cc. of filtered bouillon culture of tetanus, to which 0·5 per cent carbolic acid is added as a preservative, form the stock material. The toxic value of this culture must be such that 0·75 cc. suffices to kill with certainty an adult rabbit in three or four days. This carbolised culture is divided into four portions: (1) 20 cc. to which nothing further is added; (2) 40 cc. to which 0·125 per cent iodine terchloride is added; (3) 60 cc. to which 0·175 per cent iodine terchloride is added; (4) 80 cc. to which 0·25 per cent iodine terchloride is added. At the start 10 cc. of mixture No. 4 is subcutaneously injected into the horse, after eight days 20 cc., after another eight days 20 cc. again, and the remainder three days later. Mixtures Nos. 3, 2, and 1 are then consecutively employed, being administered gradually and at suitable intervals.

What Behring regards as essential in this method is not the mere use of cultures whose poison has been weakened by adding iodine terchloride (Vaillard uses solid iodine for the same purpose), but the subsequent employment of highly virulent cultures, or filtrates the virulence of which is unimpaired, for only thus can a degree of immunity sufficiently high to be of practical value be conferred. Cultures with unimpaired virulence had previously been employed by Emmerich and Vaillard for the production of a high degree of immunity. Behring's improved method is founded on the knowledge that the conferring of immunity is "not a sudden event, but a very gradual process." We may therefore infer "that the degree of immunity is capable of unlimited increase."

In the immunisation of the horse, even though the animal has been long under treatment, and has already been highly immunised, each fresh injection of tetanus bouillon gives rise to a reaction with fever, during which the immunising substances hitherto detectable in the urine disappear, and the urine may even contain tetanus poison (Behring and Casper, Brieger and Ehrlich). During this period no blood is to be taken from the animal for curative purposes. The former immunising value is not always regained until the eighth or tenth day, but it then undergoes gradual increase. Behring regards the appearance of the feverish rise of temperature during this period of reaction as a very favourable symptom, indicating that the poisons are being rendered innocuous in the body, and unless there be fever a further increase of immunity cannot be hoped for. The use of antipyretics in such a case is always to be avoided.

Immunisation against *diphtheria* is also best performed by the use of bouillon cultures of diphtheria attenuated by the addition of iodine terchloride. Diphtheria cultures several months old are filtered through paper, and after the addition of 5 per cent of carbolic acid they remain in contact for two to three weeks with varying quantities of iodine terchloride (0·05-0·4 per cent). Guinea-pigs, sheep, and horses are immunised by the subcutaneous administration of such cultures in gradually increasing doses (Behring and Wernicke). Each dose of diphtheria poison—treated with iodine terchloride—must induce a local and general reaction. If the reaction be insufficient the immunising effect is very slight; if the reaction be too marked, leading to emaciation, immunisation is usually frustrated. Hence, in the process of immunisation there is much care and experience required, and it is important to weigh the animal from time to time, as a permanent loss of weight must be expressly avoided. Horses are the only animals employed for obtaining diphtheria antitoxic serum, both because they stand the treatment with toxins well, and because they are well suited for the removal of blood. Blood is very easily obtained from the jugular vein, and can always be obtained absolutely pure. The venesection wound usually heals so well that blood can be withdrawn fifty to a hundred times from the jugular vein of one horse. From the blood a clear serum is easily obtained, which when injected under the skin is quickly absorbed, and even in large doses is innocuous to man and animals. The diphtheria antitoxin is often separated from the serum by precipitation and employed as a dry preparation, which requires to be dissolved before being used. If the serum is to be maintained in a fluid form, however, 0·5 per cent of carbolic acid or a small piece of camphor (Roux) is usually added.

2. *Immunisation Value.*—For the correct employment of immune sera for prophylactic and theurapeutic purposes it is, firstly, important to know that there is a definite quantitative relationship between the amount of antitoxin contained in serum and the amount of poison which the former is able to neutralise. The serum, therefore, can only bring about immunisation or cure according to the amount of antitoxic substance it contains. But in dealing with a pre-existing disease the amount of poison present in the body can never be accurately ascertained. A supply of antitoxic substance sufficient for the most unfavourable case must therefore be sought for. This is rendered possible by employing a serum of high

immunising value, which contains the antitoxin in high concentration. To obtain such a serum the animal furnishing it must have been as highly immunised as possible. Behring has proved that those species of animals which are originally very susceptible to a given poison furnish a much more potent serum than, with the same degree of immunity, do those animals which have always possessed a considerable innate resistance to the poison. The reason for this is very obvious. It is not the natural innate resistance to poison, but only the resistance which is artificially induced by immunisation, which can be transferred by means of the serum. In order, therefore, to obtain a serum with a high immunising value those animal species must be employed which are naturally very susceptible. Such is the case in the horse as regards diphtheria and tetanus.

The activity of an immune serum, therefore, does not altogether depend on the degree of immunity of the animal from which it was derived, but on its immunising value. For the estimation of the latter (*i.e.* for the standardising of serum), the method commonly used in Germany till quite recently was the one elaborated by Ehrlich, Kossel, and Wassermann. Ten times the minimum lethal dose of a toxin is mixed in varying proportions in a reagent glass with the serum which is to be examined, the mixture is made up to 4 cc. with physiological saline solution, and then injected into guinea-pigs of 250, or at most 300 grammes weight. This method yields more accurate results than the former method of injecting toxin and serum into different parts of the body. It is important to take guinea-pigs of the same weight, as otherwise the results differ. The mixture in which, under such circumstances, the toxin is exactly neutralised—there is no change whatever induced in the animal—is used in estimating the immunising value. Behring and Ehrlich first prepared a *normal toxin.* This is the term applied to a toxin solution 0·01 cc. of which suffices to kill a guinea-pig of 250 grammes weight within five days. Hence normal diphtheria toxin (DTN^1) is a solution 1 cc. of which contains the minimum lethal dose for 100 guinea-pigs, each of 250 grammes weight, *i.e.* for 25,000 grammes of living guinea-pig. *A normal serum,* or normal diphtheria antitoxin (DAN^1), is a serum 0·1 cc. of which completely neutralises the lethal action of 1 cc. of normal diphtheria toxin; 1 cc. of this normal serum contains one *immunisation unit.* A serum 0·01 cc. of which suffices to neutralise the same amount of toxin is ten times normal (DAN^{10}), and if 0·001 is sufficient the serum is one hundred times normal (DAN^{100}); 1 cc. of this latter serum, therefore, contains one hundred units. Living cultures of diphtheria bacilli used sometimes to be employed instead of toxin solution for the estimation of the immunising value of a serum, but such a method is inadvisable as the results obtained are much less accurate.

In France the ordinary mode of estimation is as follows (Roux and Martin):—The amount of serum which just suffices to protect a guinea-pig from ten times the lethal dose of toxin is ascertained. The proportion between this amount of serum and the body-weight furnishes the value of the serum. For example, "a serum whose immunising value is 10,000" signifies that an amount of serum equal in weight to $\frac{1}{10000}$th of the weight of a guinea-pig suffices to protect the animal completely against ten times the lethal dose of toxin. In the French method the body-weight of the animal is accordingly taken into account, and it is therefore not essential to use guinea-pigs of 250 to 300 grammes weight. Madsen, however, comparing the two methods, found that the German was the simpler and cheaper one.

Large numbers of immunising units (600-1500) are necessary when employing the curative serum in man. After diphtheria curative serum had become an article of commerce, it was found necessary in Germany to insist on a minimum number of immunisation units in order to protect the public from inferior preparations. The minimum first demanded was that 1 cc. of diphtheria curative serum should contain at least 100 units. Since 20th February 1895 the preparation of serum has been under state control, under which the serum is standardised, is proved to be free of deleterious substances, and the correct amount of antiseptic—carbolic acid—necessary for the proper preservation of the serum is added.

Ehrlich, the head of this serum-testing establishment, which is in connection with the *K. preussischen Institut für experimentelle Therapie* at Frankfurt-a.-M., has lately introduced a new and still more exact method of standardising serum. Diphtheria toxin and antitoxin in soluble form being somewhat variable, Ehrlich proceeds from an antitoxin dried by evaporation of the serum. This antitoxin is kept at the Frankfurt Institute in specially-constructed vacuum tubes, which each contain 2 grammes of dry antitoxin of 1700 units. Every two or three months one of these tubes is cautiously opened and its contents dissolved in 200 cc. of a fluid consisting of 10 per cent saline solution and glycerine. A serum seventeen times normal is thus obtained, and when diluted seventeen times 1 cc. contains exactly one unit. By the aid of this normal antitoxin a normal solution of toxin is then prepared by means of which again a given serum is standardised. It is by this method that diphtheria serum is now officially tested. The durability of the serum is also to some extent guaranteed by the fact that as each serum is tested a number of bottles are withheld, and their activity tested from time to time, and if it be found to diminish, all the bottles containing this serum, *i.e.* having the *same number* on them, which are still on the market are called in.

In Germany at the present time diphtheria curative serum is prepared in four different places, but it has to conform to official tests before it can be sold. More serum is prepared at Höchst-a.-M. than anywhere else, and four different forms are made at Höchst.

No. 0 contains 0·8 cc. of 250 times normal serum = 200 immunising units. An immunising dose.

No. 1 contains 2·4 cc. of 250 times normal serum = 600 units. A single curative dose for cases in which treatment can be commenced on the first appearance of symptoms.

No. 2 contains 4 cc. of 250 times normal serum = 1000 units. A double curative dose, sufficient for most cases of diphtheria when treatment begins not later than the commencement of the third day.

No. 3 contains 6 cc. of 250 times normal serum = 1500 units. A triple curative dose for very severe and advanced cases. The works at Höchst also manufacture a serum twice as strong as the preceding, *i.e.* 500 times normal. It too is obtainable in four forms, which have each, therefore, twice as many units as the preceding preparations.

The curative serum keeps very well, as a rule, for at least one year, if it be kept in a cool place and protected from light and frost. Still more durable than the fluid preparation is the dry one prepared at the works at Höchst-a.-M. by careful evaporation, and which is placed on the market under state control. It is a pure evaporated serum to which no antiseptic or other substance has been added. The preparation is in the form of fine yellowish scales or a yellowish white powder, and before being used must be dissolved in ten parts of distilled water, which takes some time. To meet official requirements 1 gramme of the dry preparation must contain at least 5000 units.

The standardisation of tetanus curative serum is essentially the same as in the case of diphtheria. Behring (and also Knorr) employed a normal tetanus antitoxin (Tet. AN), which must again be standardised by means of a tetanus toxin the nature of which is constant, and such a toxin can only be obtained by the addition of ammonium sulphate and the drying of the precipitate so obtained (Buchner). For standardisation in the *Institut f. experimentelle Therapie* at Frankfurt-a.-M. there is now used, not a dry toxin, but a ten times normal serum, which has been evaporated to dryness, for this preparation remains unchanged for years if attention be paid to the conditions under which it is kept, as above described. All tetanus serum made in Germany is subjected to official tests, as is diphtheria serum. Tetanus serum is prepared in Germany at the works at Höchst-a.-M.; the serum was formerly prepared both in fluid and solid form, but now only in the former. Each bottle contains 25 cc. of ten times Tet. AN, equal to 250 antitoxic units, suited for treating men and animals suffering from tetanus. Tetanus serum, which is protected from bacteria by the addition of 0·25 per cent *metakresol*, can be preserved for a long time, at least one year, if kept in a cool, dark place and preserved from frost.

The firm of Merck (Darmstadt) in addition supplies Germany with Tizzoni's tetanus serum, which is a dry preparation. Köhler's statistics show that it acts just as well as Behring's preparation, provided it can be injected early enough, repeatedly and freely. Before use, the dry preparation is to be dissolved in ten times the quantity of sterile fluid, and 1 cc. of the solution then contains 80,000 immunisation units, *i.e.* is able to completely neutralise 80,000 toxic units. Tizzoni's toxic unit represents the minimum quantity of filtered tetanus bouillon which can kill 1 kilo. living weight of rabbit in five to six days.

3. *The Cause of the Antagonism of Antitoxin to Toxin.*—Now there are three conceivable possibilities. Behring at first thought that there was a destruction of poison by means of the antitoxin. But if this were the case, a given quantity of antitoxin, if remaining longer in contact, would be able to gradually destroy more toxin; a mixture of antitoxin and toxin, which had at first a toxic action, would after a time have a neutral action, and would finally contain surplus antitoxin. This, however, is not the case (Roux and Vaillard, Buchner). It was also shown that a mixture of tetanus toxin and antitoxin which is almost neutral for a mouse can, when absolutely the same dose is injected, cause fatal tetanus in the guinea-pig, which is about eighteen times as heavy (Buchner). Hence in a mixture which is neutral for a mouse the poison is not completely destroyed.

This fact, however, harmonises with a second theory propounded by Roux, according to which the action of antitoxin is to be regarded as an indirect one—the antitoxin acting on the living elements of the organism and immunising them to the toxin. Roux based this view on the fact that the action of antitoxin is completely different in a healthy organism, and in one previously weakened, a fact which could not be explained by the supposition of a direct destructive action of antitoxin on toxin. In support of Roux's theory is the fact that antitoxic action is not in all cases strictly specific. For example, the blood-serum of animals which have been treated with tetanus toxin is antitoxic, not only to that toxin, but also to snake venom. The blood-serum of rabbits which have been immunised against rabies is not protective against rabies, but is markedly antitoxic to snake venom (Roux and Calmette). Similarly, the serum of animals immunised against snake venom is also antitoxic to scorpion poison (Calmette, Metschnikoff), and the immune sera obtained by means of ricin, abrin, etc., yield similar results (Ehrlich).

In spite of these facts this second theory, according to which antitoxin and toxin are *physiological antagonists*, cannot be regarded as correct. There is more probably a direct combination of antitoxin and specific toxin, whereby the action of the latter on the organism is arrested, as was first shown by Ehrlich and then by Knorr. But here too it is necessary to determine more definitely the nature of this combination which Ehrlich regards as a simple chemical one like that between acid and base. Recent investigations carried out by Ehrlich with ricin and antiricin, by H. Kossel, Camus and Gley, and Kanthack, prove beyond doubt that antitoxin and toxin directly affect and combine with each other. Possibly, however, there is not a simple chemical combination, but a sort of absorptive action, such as we see in connection with staining agents. This latter supposition is as a matter of fact by no means improbable, for it is only thus that a series of facts ascertained by A. Knorr can be explained. There is no doubt that toxin and antitoxin generally combine in proportionate amounts, so that if one unit of toxin be neutralised by a given quantity of antitoxin, one hundred times as much antitoxin is able to combine with one hundred units of toxin. Yet there are deviations from this rule. One striking fact is that a concentrated mixture of toxin and antitoxin differs from a dilute mixture in regard to neutralisation. For instance, Knorr, using the same toxin and antitoxin in each case, obtained the following results:—

250,000 units toxin + 250,000 units antitoxin = 0
2,500 ,, ,, + 2,500 ,, ,, = } Mild tetanus
25 ,, ,, + 25 ,, ,, = }

Again, the length of time the toxin and antitoxin have been in contact before being injected is of importance in determining their combination; for example, a mixture of 2500 units toxin + 2500 units antitoxin immediately after being prepared was injected into an animal, and death from tetanus occurred three and a half days later. The same mixture if kept for two hours caused only slight disease, and after being preserved for twenty-four hours it became inactive. Such combinations vary very greatly according to the nature of the poison in question. The subject has only become comprehensible since Ehrlich showed how greatly the constitution of various preparations of one and the same poison may differ, according to the amount of toxones, toxoids, etc., they contain, *i.e.* the amount of modified toxins which are non-poisonous, and yet which combine with antitoxin. These differences in the various toxic preparations, therefore, do not help to solve the problem. Now, if there were merely a chemical combination of toxin and antitoxin, it would necessarily follow, when dealing with one and the same solution of toxin and antitoxin, that the degree of concentration would be of little importance. In point of fact, however, this is not so. Knorr, for example, working with his standard toxin and standard antitoxin, obtained the following results:—

	Toxin.		Antitoxin.		
1.	250,000 units	+	250,000 units	=	0
2.		+	225,000 ,,	=	Mild disease.
3.		+	200,000 ,,	=	Death in 4 to 9 days.
4.		+	150,000 ,,	=	Death in $2\frac{1}{4}$ to 4 days.
5.		+	125,000 ,,	=	Death in $1\frac{1}{2}$ to $2\frac{1}{2}$ days.
6.		+	100,000 ,,	=	Death without delay.

Thus in the fourth experiment 100,000 units of toxin were reckoned as being in excess. *Their physiological action, however, is merely equivalent to that of* 15 *units of free toxin,* and to compensate the toxic effect of mixture No. 4, and to render it neutral again, there are not 15 units of antitoxin but 100,000 units required.

The inference is that in a concentrated solution a unit of antitoxin may sometimes combine loosely with more than one unit of toxin, so that the latter's toxic action is greatly diminished. This too is the real cause of the results experimentally obtained by Buchner, who, as already noted, found that a mixture of tetanus antitoxin and toxin which is almost neutral for a mouse, can produce fatal effects in guinea-pigs. The loosely combined portion of the toxin has an action which varies in degree according to the specific susceptibility to toxin of the different animal species. The exact nature of the combination between antitoxin and toxin is still unknown, but the fact that there is a combination cannot be doubted. The antagonistic effect of antitoxin on toxin is due to this combination, which is therefore one of the most important facts in the realm of immunity.

Another no less important question is that of the *nature and origin* of antitoxins. Behring considers them to be products of the animal body, formed in consequence of the feverish reaction attending injection of toxic filtrates. This, however, is not invariably so, for, according to Metschnikoff, the crocodile (Alligator Mississippiensis) is distinguished from all other animals by the rapidity in the formation of antitoxin, *although there is no feverish reaction.* Twenty-four hours after the injection of tetanus toxin and six days after the introduction of cholera toxin in solution, the alligator's blood becomes antitoxic to these poisons to which it is very insusceptible. Yet, when full-grown alligators are contrasted with young crocodiles and turtles, it is remarkable that both the latter form antitoxins very slowly if at all, and are yet very insusceptible to tetanus toxin, and can stand enormous doses of it.

Ehrlich has recently furnished, by means of his "*side-chain theory*," a definite and in many respects a very instructive explanation. According to this theory the general action of toxins is as follows:—Each molecule of toxin possesses two different chemical groups; one is "haptophorous," and by means of this the toxin becomes firmly bound to any cell protoplasm of the organism for which it has a chemical affinity, and a "toxophorous" group by which disturbance is then induced in the protoplasm in question. The toxin does not become bound to the "functional centre" of the cell protoplasm, but to a side chain of the latter. Hence the name of the theory. If the action of the toxophorous group of toxin molecules causes disturbance in the cell protoplasm, and if certain side-chains are consequently destroyed, the latter, it is said, are re-formed by the regenerative efforts of the organism. If this process be of considerable intensity, there may be excessive production of side-chains, the excess is transferred to the blood, and possessing, as already mentioned, a chemical affinity for the haptophorous group of the given specific toxin, acts as antitoxin. This ingenious theory explains in a relatively simple way the remarkable fact that the organism responds to the introduction of toxin by the formation of a specific antitoxin. The supposition that the action of toxin in the body is dependent on the chemical affinity of toxin for definite cell-protoplasm is undoubtedly correct. The theory, however, is open to hostile criticism.

Another theory, held by Buchner and Metschnikoff, is that antitoxins represent in general non-poisonous modifications of toxins, which are elaborated by certain cellular elements of the body and transferred to the blood. Space forbids a detailed account of the facts on which this view is founded, but it may be said that this view harmonises better than does the side-chain theory with recent observations which indicate that the organism reacts with the formation of antitoxin, not only after the introduction of bacterial toxins, but that it reacts similarly on the introduction of the red blood corpuscles of any other species of animal, forming a specific anti-substance which passes into the blood; the same occurs on the introduction of foreign epithelium, leucocytes, spermatozoa, etc. The elaboration of anti-substances must therefore be regarded as a universal mode of reaction against foreign cells and their products, and not merely against toxins.

4. *Transference of Antitoxin by Inheritance and Suckling.* (*Milk.*)—Ehrlich showed that in mice rendered tolerant of ricin or abrin the immunity is transmitted to the offspring from the mother, but not from the father, and he found that this tolerance of poison is only in part transferred from the mother's serum, but is mainly due to a subsequent transference of antitoxins by means of suckling. He proved this fact by the "exchange or wet-nurse experiment," in which the newly-born offspring of a highly-immunised mouse and that of a control mouse were respectively suckled by the different mothers. The offspring of the highly-immunised mouse, on being suckled by a normal control animal, possessed only a very slight degree of immunity after twenty-one days, in consequence of the excretion of the antitoxins originally inherited from the mother, whilst the immunised mother was able to transmit the antitoxin by her milk, and a marked and progressive immunity was thus conferred to the offspring of the control animal. The transference of immunity or tolerance of poison to the offspring by means of the milk was subsequently demonstrated in tetanus (Ehrlich), and it was shown that the subcutaneous injection of the milk of goats which have been immunised against tetanus confers protection from tetanus (Brieger and Ehrlich). Antitoxin has been successfully precipitated from milk and prepared in a dry concentrated form. The antitoxins contained in milk are absorbed from the intestinal canal without undergoing change mainly because of their marked stability. Ehrlich shows that the maternal milk may also be serviceable for the transference of antitoxin to the human infant, and indicates that it is possibly owing to this fact that a number of infectious diseases (mumps, scarlatina, measles) so seldom occur during the first year of life.

5. *Employment of Immune Sera for Immunisation and Curative Purposes.* (*Prophylactic Employment and Serum Therapeutics.*)—Two forms of immune serum can be differentiated: the antitoxic serum, and the serum the action of which is only anti-bacterial or bactericidal. Results of real value have hitherto only been obtained with antitoxic serum, but brief mention must be made of the results gained by the use of bactericidal sera. In diphtheria and tetanus more especially it has long since been proved experimentally that a cure may be effected by antitoxic serum even after the disease has commenced (Behring, Kitasato, Wernicke, Knorr), the explanation of the curative effect being the above-mentioned combination of antitoxin and toxin. But a relatively large amount of antitoxin is required if treatment is to be successful, and the longer the time elapsed since the commencement of the illness the more antitoxin is necessary. In the late stages of the disease the intoxication is so great that large doses of antitoxin are ineffectual. Under such circumstances an antitoxin of the highest possible concentration is of great value, and it has often been successfully prepared by precipitating the antitoxin from the serum and subsequently making a concentrated solution. Another method of gaining the same end is to freeze and then thaw the serum, thus separating water and proteid, a process which may be promoted by centrifugalisation.

Diphtheria.—Antitoxic diphtheria serum is but seldom used for immunisation. The usual dose is 200 units, and if danger of infection persists the inoculation is to be repeated about fourteen days later, because of the rapid excretion of the antitoxin from the body. The use of larger doses of serum is not so suitable, because the more concentrated the antitoxin in the blood the more rapid is the excretion. It is difficult to furnish absolute proof of the protection man derives from these inoculations; to do so would require many statistics. The results gained by Löhr and Slawyk in the children's clinic at the Berlin Charité are interesting. By immunising all the children, including those newly admitted, they succeeded in checking the spread of diphtheria in the clinic, as could not be done otherwise, whilst mere immunisation of those children in beds adjoining that of a diphtheria case had failed to do so. The immunising dose was usually 200 units, which was borne without risk.

The *therapeutic* application of diphtheria serum is based on the experimental studies of Behring and Wernicke (1892) on the recovery of guinea-pigs after subcutaneous inoculation with diphtheria bacilli in such doses that the animals would have died in three or four days had they not been treated. If treatment were commenced immediately after infection, 1½ to 2 immunising doses were required to effect a cure; eight hours after infection, 3 immunising doses; and twenty-four to thirty-six hours after infection, 8 immunising doses were requisite. After small doses of diphtheria bacilli, when the disease in the guinea-pig runs a slower course, the curative action can also be recognised in the local changes at the seat of injection, for the serous exudation which has been formed quickly disappears. If infection has occurred some considerable time previously so that the disease is already advanced, large doses of antitoxin cannot save the animal. The disturbances, which are already in existence in the various cells of the body, can no longer be influenced by the antitoxin. The action of diphtheria serum can also be experimentally shown in the case of a true superficial diphtheria produced by inoculation of diphtheria bacilli on the vagina, trachea, or other mucous membranes of animals. The false membrane so formed becomes detached as the animal recovers (Roux and Martin). The action of the serum is only antitoxic, not anti-bacterial; but as the diphtheria bacilli do not spread into the body from the seat of inoculation, the organism, after the use of curative serum, gains time to gradually overcome the bacilli by means of its natural protective powers.

For use in medical practice the rule is to inject large doses as soon as possible, giving at least 1000 units, in severe and urgent cases 1500 to 3000 units, or even more. The antitoxin must be regarded as absolutely innocuous; any by-effects, such as skin eruptions or pains in the limbs, which sometimes occur, are due solely to the serum, normal horse's serum being able to cause similar symptoms. The injections are made with a sterilised syringe under the skin of the thigh, or between the shoulders, with strict attention to asepsis. The serum must be clear or somewhat opalescent, it should not show cloudiness. Statistical proof of the curative action of the serum has been furnished from all sides; for example, Roux's experience is that since the introduction of curative serum the mortality has fallen from 50 per cent to 26 per cent. The decrease of mortality according to Heubner is from 44·3 to 21·1 per cent, according to Baginsky from 48·4 to 15·6, according to Ranke from 57 to 18·8, and according to Monti from 34 to 22 per cent. The joint inquiry of the *Deutsche medicinische Wochenschrift* showed that in 5833 cases treated with serum the mortality was 9·6 per cent, whilst it was 14·7 per cent in 4479 cases not treated with serum. Statistics also prove that the sooner treatment is begun the more favourable are the results. The following are the results published by the German Imperial Board of Health, the investigations dealing with 204 hospitals in Germany for the period from April 1895 till March 1896:—

Total number of cases	9581	
Total mortality	15·5	per cent
Mortality when treatment began on the 1st day	6·6	,,
,, ,, ,, 2nd ,,	8·3	,,
,, ,, ,, 3rd ,,	12·9	,,
,, ,, ,, 4th ,,	17·0	,,
,, ,, ,, 5th ,,	23·2	,,
,, ,, after 5th ,,	26·9	,,

Behring's experience is that those cases of diphtheria in which treatment with a single curative dose (600 units) is begun within forty-eight hours of the commencement of the disease will have a mortality of less than 5 per cent.

Many doctors, moreover, maintain that the clinical course of disease is favourably influenced by the curative serum. There are those, however, who hold a contrary opinion, which is perhaps due to the fact that in diphtheria there is often a mixed infection with streptococci, and that the latter are not influenced by the serum. But the majority of observations indicate that it is more especially in laryngeal diphtheria that injections of serum induce the most favourable effect, the signs of stenosis usually disappearing rapidly after the injections. It cannot be denied that post-diphtheritic paralyses are as numerous since the introduction of serum treatment, or that they have become more frequent still; but this is possibly due to the fact that a greater number of severe cases recover now than formerly. It is often said that curative serum causes albuminuria. According to the statistics of the German Imperial Board of Health, two-thirds of the cases treated with serum had no albuminuria, although it is by no means of rare occurrence in diphtheria. Unpleasant by-effects, such as skin rashes of an urticarial character, joint pains and swellings, can doubtless be caused by the diphtheria curative serum. The cause thereof is not the antitoxin, but the serum, which even when normal can induce such conditions in persons predisposed thereto. Such effects are in no way dangerous, but it would be well if the antitoxin could be separated from the constituents of the serum and prepared in a pure form.

Tetanus.—The prophylactic use of serum is of greater importance in tetanus than in diphtheria. Immunisation against tetanus was carried out by Nocard (1895-97) in 2373 horses and 332 other large animals. None of these succumbed, whilst during a similar period of time and under the same conditions there had previously been 191 cases of tetanus in horses and 68 cases in other animals.

Immunisation, therefore, for which small doses costing but little are sufficient, is advisable previous to any operations on the horse when cleanliness is a matter of difficulty, and also after such wounds as are known to be often followed by tetanus. In man, too, tetanus serum as a prophylactic agent is very useful after severe contused wounds contaminated with soil, dust, manure, etc. The duration of the immunity conferred is comparatively short except when horses are inoculated with tetanus serum obtained from horses.

Behring and Knorr, injecting tetanus toxin into animals, have experimentally investigated the possibility of curing tetanus with serum. If only the minimum lethal dose be given it may be possible to save the animal by the introduction of serum even a considerable time afterwards. If, however, the intoxication be induced by a larger quantity of toxin—for example, 100 times the minimum lethal dose—the amount of antitoxin subsequently required becomes much greater. Even a quarter of an hour after the injection of toxin it is necessary, in order to effect a cure, to use 100 times as much antitoxin as would have sufficed to neutralise the same quantity of toxin had they been mixed *in vitro* prior to injection; and very soon, long before any tetanic symptoms appear, it becomes impossible to save the animal. It is much easier to cure animals after they have been infected with tetanic spores, as occurs in natural infection, than when they have been poisoned with prepared toxin (Kitasato, Knorr), and such experiments prove that the antitoxin may really effect a cure even though symptoms of the disease have appeared. The difficulty of curing an advanced case of tetanus by means of serum is probably due to the fact that the toxin is soon absorbed by and bound to the tissues of the body, and the neutralising effect of the antitoxin thus inhibited. It has been shown in rabbits that to prevent the onset of tetanus a dose of serum, if given twenty-four hours after the injection of toxin, must be twenty-four times as great as that which suffices when given at the same time as the toxin (Dönitz). This indicates that even in the first few hours the greater part of the toxin has combined with the tissues of the body, for otherwise subsequent neutralisation in the blood would be possible by means of the antitoxin. In spite of this combination a sufficient amount of serum may even yet effect a cure, hence we must assume that antitoxin when present in abundance is able to withdraw the loosely combined toxin from the tissues, or to neutralise it while still there.

The results hitherto gained in large animals, and especially in horses, are indeed not very favourable, but would undoubtedly be better were treatment begun sooner and larger doses given. In man the difficulties in the way of successful treatment of tetanus with serum are still greater, and there is no statistical proof that the results have improved since antitoxic serum was introduced. This is obviously due to the fact that by the time the first tetanic symptoms appear, the toxin has already become combined in the nervous system, and that the disturbances there can no longer be influenced by the antitoxin. The great importance of the prophylactic use of tetanus serum must therefore be again emphasised, for in this case the antitoxin present in the body may be confidently expected to neutralise the toxin on its first appearance in the blood, and *before* it has combined with the tissue elements. The attempts to render the toxin more active by means of intravenous injection, or by direct introduction beneath the dura mater (Roux), have hitherto not yielded results of practical value.

Plague.—Plague serum is prepared by treating horses with increasing doses of living or killed plague cultures, and usually by means of intravenous injections (Yersin, Roux, Wladimiroff). Each injection causes fever and signs of a general reaction. A serum is finally obtained which in even somewhat small doses can protect other animals against infection with plague. At the Pasteur Institute in Paris the serum is standardised by estimating the smallest dose which can protect mice against infection twenty-four hours subsequently with the minimum lethal dose of plague bacilli. Yersin in 1897 obtained favourable results with such a plague serum, for of 500 inoculated individuals in a plague-stricken district only 5 sickened and 2 died. In 2 of these 5 cases the disease occurred so soon after the inoculation that infection had probably already occurred, and they should not be counted. The symptoms in the other 3 cases ensued twelve to forty-two days after inoculation, *i.e.* probably after the protective effect had expired, for we may conclude from animal experiments that the protection conferred only lasts ten to fourteen days. The experimental researches of the German Plague Commission on the immunising value of Yersin's plague serum demonstrated that by means of 10 cc. monkeys can generally be immunised against several times the minimum lethal dose. There is, however, a form of gray monkey which has a special susceptibility to plague, and in which the protective action of the serum is of no avail, and it therefore appears doubtful whether effective protection can be obtained for the human subject.

The above-mentioned short duration of the protection conferred by plague serum is, however, a practical drawback, which does not exist in the active immunisation with killed plague cultures. In the most urgent cases a combination of active and passive immunisation—injecting killed cultures and plague serum together—would perhaps be advisable.

On the therapeutic application of plague serum, reports were given by Yersin from India (1896 and 1897), which, however, require further confirmation. The German Plague Commission had the opportunity in Bombay of studying the results of serum treatment in a series of 26 individuals, whose ages varied from 1½ to 60 years. They were all recent cases of simple bubonic plague, in whom the prognosis would in any case have been favourable, so that although the mortality in these cases was equal to 50 per cent, and therefore appears more favourable than the usual average mortality (80 per cent) from plague, it is no proof that the serum had a curative effect.

Both the German and the Russian Plague Commissions investigated the curative effect of the serum in animals. In one breed of monkeys, which are not very susceptible to plague, a cure was effected by means of 10 cc. of serum even six and twelve hours after infection with a lethal dose of plague cultures. If the administration of the serum were further delayed the result was uncertain or negative. The results will undoubtedly be better if a more potent plague serum can be prepared. The serum is not, or only to a slight degree, antitoxic; its action is mainly a specific bactericidal one, just as in animals treated with cholera and typhoid bacteria. The condition in plague is more a bacterial infection than an intoxication, as is also the case in anthrax, for example. Roux, however, believes that the antitoxic action of plague serum is dependent on its mode of preparation, and that the serum contains most antitoxin when obtained by the intravenous injection of living plague bacilli.

Cholera.—In cholera, as in plague, few definite results of practical value have been obtained. It is indeed easy to obtain a specific bactericidal serum by treating animals with cholera vibrios. But the value of such sera for immunisation appears to be slight; large doses would have to be used, and in spite of them the protection would be of short duration. Behring and Ransom, Roux and Taurelli-Salimbeni attempted to isolate a toxin from cholera vibrios, and to obtain an antitoxic cholera serum by treating horses with this toxin. It is claimed that this serum protects not merely against cholera intoxication, but also against the living cholera virus. No use has as yet been made of this serum on the human subject. In man, therefore, active immunisation by killed cholera cultures appears as yet to be the only practical method of proved value.

The *therapeutic* value of cholera serum is naturally still more doubtful, especially when the rapid course of the disease is borne in mind. Attempts to treat cholera patients with the blood serum of those who had recovered from the disease have been very indecisive.

Typhoid fever.—By treatment with killed cultures (R. Pfeiffer), a serum is obtained which is specifically bactericidal in a moderate degree, but not antitoxic, and therefore unsuited for therapeutic purposes in man. For intoxication is the main feature in typhoid, and an antitoxic serum would therefore be required.

Pfeiffer's results have been corroborated by many writers. Beumer and Peiper obtained a serum from sheep by a method essentially the same as Pfeiffer's, but it had no curative action in typhoid. Apparently good results were obtained by Klemperer and Levy, who got the serum from dogs, but the typhoid cases in which it was employed were mild, so that the observations are not decisive.

Pneumonia.—Attempts to obtain an immune serum and to employ it in pneumococcal infection in animals were formerly made. G. and F. Klemperer were the first to employ the serum of rabbits, which had been treated with pneumococci, in pneumonia in the human subject, having previously proved that such serum can effect a cure in pneumococcal infection in rabbits. They sought to prove the etiological identity of these two diseases by showing that the serum of persons convalescing from pneumonia can also bring about recovery from pneumococcal infection of rabbits. An injection of 4-6 cc. of the serum appeared to influence the course of pneumonias favourably, in that the temperature often fell and remained down. Observations were, however, only made on six cases, so that no definite opinion can be given. The action of the serum is probably bactericidal and not antitoxic (Issaëff and Bonome).

Streptococcal Infections.—Behring, Knorr, and others had worked at immunisation against streptococci, but without definite success. In 1895 Marmoreck claimed to have obtained an antistreptococcus serum from rabbits, and afterwards from horses. Favourable reports as to its value for the human subject were soon published in France. For immunisation, horses are subcutaneously inoculated with gradually increasing doses of cultures of streptococci, which are highly virulent for rabbits. Each injection is given only after the complete disappearance of the signs of reaction, which are very intense. The serum of such animals is then claimed to exert a protective and curative action in streptococcal infections. This was experimentally confirmed by Bordet, but others (Aronson, Petruschky) obtained absolutely negative results. The apparently favourable results attained with this serum in erysipelas and puerperal fever are too scanty, and the use of the serum does not sufficiently explain the direct change induced in the temperature, etc.

It was afterwards asserted by Denys and others that a given streptococcus can induce immunity and furnish a protective serum only against streptococci of the same stock, and that a number of streptococci of different stocks must therefore be simultaneously made use of when immunising. But it was also shown that streptococci of different stocks which are virulent only for animals may be quite unsuitable for the production of a serum which is to be employed in man. Hence Tavel for the immunisation of horses only employs such streptococci as have been obtained directly from the human subject. There is but little known of the clinical results obtained by such serum. But on the whole it is improbable that a potent antistreptococcus serum will be obtained, because the serum of persons who have recovered from a streptococcal infection has no power of immunising against the same streptococcus, and again because streptococcal infection in man confers no protection, or even predisposes to a subsequent attack, as in erysipelas of the face, for example. The apparent benefit from the use of such serum in many clinical cases is possibly due to an increase of natural resistance induced by hyperleucocytosis.

Staphylococcal Infections.—Animals can be rendered immune to staphylococci by giving them increasing injections of cultures of staphylococci. Viguerat rendered goats highly immune by this method, and then used their serum for therapeutic purposes in staphylococcal infections in man. But the results recorded cannot be regarded as proof of a curative action. Subsequent investigations by Petersen have indeed confirmed the fact that animals can be immunised against staphylococci, but the value of the immune sera obtainable is too slight to admit of the serum being of practical value for the human subject. According to Petersen these sera are anti-bacterial and not antitoxic.

Intoxication with Snake Venom.—According to Calmette, the various varieties of snakes have different venoms, and in a given snake the virulence varies according to the length of time the snake has fasted, becoming gradually and steadily greater. Rabbits, guinea-pigs, donkeys, and horses can gradually be rendered immune to what is otherwise a lethal dose of venom by treating them at first with small and later with increasing doses of venom. Their serum then possesses antitoxic and curative powers, both of which, according to Calmette, are tested on rabbits. A rabbit is first given 2 cc. of serum, and five minutes later the minimum lethal dose of snake venom is injected intravenously. Without the serum the animal would die in 15 to 30 minutes; if the serum be potent the animal remains healthy. To test the curative value, an analogous method is employed. The lethal dose is injected into a vein of a rabbit, and five minutes later, when the venom is commencing to take effect, 4 cc. of serum are given intravenously. The animal should then recover in a short time. All the serum prepared by Calmette has to conform to these two tests.

It is important to know whether the venoms of the various varieties of snakes are essentially identical. Cunningham differentiates two forms, *i.e. blood poisons*, such as the venom of cobra capella, and *nerve poisons*. Calmette holds a contrary opinion, and refers to the fact that his serum has been used with excellent results for the bites of quite a number of different snakes. His serum is even claimed to be antitoxic to scorpion poison (a point corroborated by Metschnikoff), although the latter is undoubtedly different from snake venom.

Owing to this fact and to others previously mentioned, we may conclude that the antitoxic action is not so markedly specific in the case of these venoms as in the bacterial poisons. The former also differ from the latter in being more resistant to high temperatures, undergoing no change at 80° to 90°. Calmette's assertion, that the serum of an animal which is rendered highly immune to a given snake venom also confers protection against the venoms of other varieties of snakes, is accordingly by no means improbable.

Calmette's results have been in the main confirmed by Fraser, who succeeded in obtaining a very potent anti-venom serum from the horse. According to Fraser, 200 to 300 cc. are required to save a man after being bitten. The serum has hitherto not been sufficiently used in India to permit of definite conclusions being drawn. Fraser states that the bile of venomous snakes may also be antitoxic. By precipitating with alcohol, he obtained an antitoxin of high curative value, which, even when given thirty minutes after a lethal dose of cobra poison, was able to save the animal, the proportionate weight of antitoxin employed to body-weight being 0·01-0·07 : 1000. Calmette regards this phenomenon as not being due to the action of an antitoxin, but considers the bile substance as an unusually active stimulant, yet such an explanation does not diminish the practical significance of the fact.

In every form of snake bite Calmette recommends the employment not only of antitoxin, but also of chloride of lime solution, freshly prepared, to be used as a wash and also subcutaneously injected, after a firm ligature has been placed round the limb; 20 to 30 cc. of antitoxic serum are to be injected under the skin of the abdomen.

Tuberculosis.—Behring has striven for many years to obtain an antitoxic serum for tuberculosis. His aim was to first prepare a potent toxin from tubercle bacilli and then to immunise animals therewith. He succeeded in preparing a toxin the toxic activity of which is considerably greater than that of Koch's tuberculin. Tuberculous cattle were cured by gradually increasing doses of Behring's toxin, and simultaneously rendered immune, so that they were finally able to withstand a dose of toxin lethal to healthy cattle. But the serum of such animals possessed only a slight protective power against Koch's tuberculin. In regard to the production of a tuberculosis antitoxic serum, Ransom hoped to get better results from birds, but none of practical value have been recorded.

The employment of birds was mainly suggested by the fact that the mere use of the serum of horses and cattle, not only of those which have been inoculated, causes fever in phthisical patients, which is not the case with the serum of most birds.

At an earlier date Maragliano stated that he had obtained an antitoxic serum by treating the larger animals, and horses more particularly, with tuberculin toxin. The simultaneous injection of tuberculin and this serum is said to cause neither local nor general reaction. This serum has been much used by Maragliano and other Italian physicians, but there is no definite proof of it having been employed with success. It is on the whole very doubtful whether a true antitoxic serum can be prepared in the case of tuberculosis, for in this disease the toxic action is not very pronounced, and the experience of most men is that recovery does not confer immunity.

Meat Poisoning.—The B. botulinus is frequently an etiological factor, and its toxins are somewhat analogous to those of diphtheria and tetanus. After treating animals with filtrates of bouillon cultures of B. botulinus, they may furnish an antitoxic serum which has both a prophylactic and curative action (Kempner).

Swine Erysipelas.—Lorenz's method of protective inoculation is the most successful and most frequently used in Germany. It is based on a judicious combination of active and passive immunisation. The possibility of immunising against swine erysipelas by either method had previously been pointed out by Emmerich. Yet passive antitoxic immunisation alone has the disadvantage of conferring a protection of too short duration, whilst simple active immunisation with living cultures implies the risk of inducing serious disease. What Lorenz aimed at was, firstly, to render the animals somewhat immune by injections of serum, and then a few days later to inject ¼-1 cc. of a living bouillon culture. Protection is thus conferred and lasts for about five months. If this be not considered sufficient, another injection of 0·5-2 cc. of living culture must be made about a fortnight later, when the resulting protection lasts for about one year. Before the end of that time further injections may be given and the protection indefinitely prolonged. Lorenz has of late obtained his serum from horses, and he treats it so that it shall keep.

Of 4540 swine immunised by this procedure in Germany in 1896, only two died, and that too seven months after treatment. According to Voges and Schütz this procedure is best adapted for the finer breeds of swine, whilst Pasteur's method, as already mentioned, is more suitable for the ordinary breeds.

Cattle Plague (*Rinderpest*).—The investigations of R. Koch, Kolle, and Turner show that there are two main forms of protective inoculation for cattle plague, the virus of which is still unknown. The one method is the use of the bile of animals which have died of cattle plague. The injection of 10 cc. of such bile confers, about ten days later, an immunity which lasts for several weeks, and which according to Koch and Kolle is an *active* one, the bile containing the attenuated virus. Compulsory inoculation with bile was attended, in Basutoland for example, with the following results: of 100,000 animals 70,000 remained alive, whilst all the uninoculated herds died. Kohlstock modified and improved this method; subsequent to inoculation with bile he injects 1 cc. of virulent cattle plague blood.

As the bile of 3-7 oxen is required for the immunisation of 100 cattle, the *combined* use of serum and cattle plague blood is as a rule better than the "bile method." Cattle which have recovered from the disease are given increasing injections of cattle plague blood. They eventually become highly immune, and furnish a very potent serum which may even effect a cure after the disease had commenced. 15-40 cc. of such serum are injected into one flank, 1 cc. of virulent cattle plague blood being injected at the same time into the other flank (simultaneous method). The results appear to be very satisfactory; of 9007 inoculated animals only 178 (1·4 per cent) died of cattle plague, and moreover the immunity thus obtained lasts for some months. This latter method is not only the more effective, but also the cheaper of the two, since a highly immunised animal can furnish serum for 300 others. Yet the "bile method" is of service when a herd is attacked with the disease, and there is then a sufficient supply of the vaccine.

CONCLUSION.—The latest discoveries in connection with immunity are characterised by the *specific hæmolytic* actions which Bordet was the first to investigate in detail. If a definite quantity of rabbit's blood be introduced into the peritoneum of a guinea-pig, the red blood corpuscles of the rabbit are somewhat slowly dissolved and eventually disappear completely. If, however, the same animal be given such injections repeatedly, its serum acquires a *specific hæmolytic* action on the rabbit's red blood corpuscles, so that they are much more rapidly destroyed than they would be in the serum of a normal guinea-pig. But the increased hæmolytic power of the serum has reference only to the red blood corpuscles of a rabbit, and not to those of any other animal. The condition is therefore analogous to the specific bactericidal actions. In a similar manner the serum of a rabbit may acquire a specific hæmolytic action on the red blood corpuscles of the ox or the dog, after being treated with the blood of the ox or the dog respectively. The same rule probably holds good for the red blood corpuscles of all mammals in relation to all the different species of mammals.

Analogous conditions have been demonstrated for other forms of animal cells (ciliated epithelium, leucocytes, spermatozoa) by v. Dungern, Metschnikoff, and others, for after treating an animal with such cells its serum acquires a specific destructive power on similar cells. Such specific treatment, which incites the elaboration of specific protective substances in the body, can therefore be carried out in an indefinite number of instances.

Bordet proved that specific hæmolytic action is similar to specific bactericidal action in being due to the *combined action of two different substances,* on the one hand that of a specific anti-substance which is preserved at 65° C., and on the other that of the non-specific alexin which effects the dissolution of the hæmoglobin. The process in detail is that the specific anti-substance is attracted and bound to the blood corpuscles in question (Ehrlich and Morgenroth), which thereby become predisposed to the solvent influence of the alexin. The analogy to the specific bactericidal action is therefore complete; in the latter instance the specific anti-substance (agglutinin) which is preserved at 65° C. becomes bound to the specific bacteria (Gruber, Hahn, and Trommsdorff), which are thereby rendered susceptible to the action of the alexins.

But we also know that the antitoxins—the specific substances of tetanus, diphtheria and other sera, which are preserved at 65° C.—neutralise the specific toxins by forming a sort of loose combination with them, and that here at any rate the action is in no way due to the alexins, because there are in this instance no foreign living cells.

In consequence of all this, the essence in all cases of *specific* immunity consists in the fact that *the organism is protected by the formation of a specific definite anti-substance or antitoxin which is preserved at* 65°, *and which has a peculiar attraction for any foreign invader* (toxin, bacteria, red blood corpuscles, etc.), *and that what is thereby effected is a neutralisation* (of poisons), *or an alteration permitting the protective action of the normal alexins to be displayed* (in the specific bactericidal, specific hæmolytic actions, etc.).

The condition in which the organism because of prolonged administration of poison becomes accustomed to it, or as is commonly said becomes immune, is not in every case due to the formation of antitoxin. According to recent researches by Edw. Faust the increased tolerance of morphia is not due to antitoxin formation, but to an increased capability of the body of destroying morphia. *True immunisation with the formation of antitoxin is only possible in the case of substances without definite chemical constitution* (proteids or their allies), and not in the case of simpler molecules.

Lastly, we have once more to note that the phenomena of immunity are *in part to be included in the realm of "specific immunity,"* and in part in that of "natural immunity" for which we have employed the term "natural resistance." "Specific immunity" and "natural resistance" are in theory totally distinct conditions, but in practice they very frequently, almost always, act in combination in one and the same individual in warding off the infective agent. An infective process cannot be correctly understood without taking into consideration both forms of immunity and their variations. The difference between the two forms of immunity is best characterised by their active substances: the alexins of natural resistance are destroyed at 60°, vary according to the animal species forming them, and are not attracted to the toxins, bacteria, etc. The antitoxins and anti-substances of specific immunity do not derive their character from the animal species forming them, but from the special form of toxin, bacteria, etc., which were employed in treatment, and they are specifically attracted to the toxins, bacteria, etc., which are attacking the organism. Every organism possesses a certain degree of natural resistance at the moment when the disease commences, a resistance which, because of the attempts at protection, will usually increase during the disease, but which may decrease, whilst anti-substances are formed and brought into action. An infective disease, therefore, as a rule represents a very complicated process when regarded from the standpoint of immunity. The analysis of the individual factors of this combined process falls to the task of clinical medicine, and it is to be hoped that in the course of time not merely theoretical explanations, but also points of practical importance will be discovered.

LITERATURE.—A record of current literature is contained in :—BAUMGARTEN. *Jahresbericht d. path. Mikroorganismen.*—KOCH and FLÜGGE. *Zeitschr. f. Hyg. u. Infects.*—*Centralb. f. Bakt. u. Parasit.*, Abthl. I.—MALY. *Jahresbericht ü. d. Fortsch. d. Thier-Chemie.*

Impetigo.—The term impetigo (*ab impetu*) is applied to an eruption, or group of eruptions, characterised by the evolution of small superficial epidermic vesicles, vesico-pustules, or pustules of the kind formerly described as psydracious, and distinguished clinically from the larger ecthymatous pustule, the acneiform perifollicular pustule with indurated base, and from the sycosiform pustule. Not all pustular eruptions are, however, impetigo. There is a rare, primary, pustular disease of a totally different nature known as *impetigo herpetiformis.* In all the vesicular and bullous eruptions the contents of the lesions may become puriform, *e.g.* vesicular eczema, dermatitis herpetiformis, herpes. On the other hand, a host of pruritic eruptions may be complicated by pus formation following scratching, *e.g.* eczema, animal parasitic diseases, prurigo, lichen urticatus, prickly heat. As the pus formation thus secondarily complicating eruptions is probably due to microbes similar to those producing the impetigo under consideration, it is evident that the subject is a very complex one. It is at the present moment being subjected to rigorous investigation at the hands of bacteriologists.

The dismemberment of the Willanean groups *porrigo* and *impetigo*, and the transference of the constituents to the pustular or impetiginous phases of eczema, left one variety to be dealt with, characterised by contagious and auto-inoculable "favous pustules" (*porrigo favosa* of Willan and A. T. Thomson, *impetigo favosa* of Bateman), and even this was wrongly included under eczema by Hebra, and confounded by Bayer and Biett with favus on account of the name. Startin, senior, whose name is frequently mentioned in connection with the history of the eruption, identified this affection with one he had christened *porrigo simplex* (*Medical Times*, 1846), and Devergie in France also recognised its contagious nature. In 1864 and 1869 Tilbury Fox attracted general attention by his papers on *impetigo* or *porrigo contagiosa*, and thenceforward the literature multiplied. Fox described a *vesicular* eruption, primarily of systemic origin and tending to run a definite course, but kept up by secondary auto-inoculation. He proved the inoculability experimentally, and was corroborated by van Harlingen. This definite picture was somewhat blurred by the further observation that similar phlyctenæ might arise out of and around vaccination crusts, about cuts and like injuries, and that it might complicate eczema, scabies, and other affections, and *vice versa.* He expressly states that pediculi were absent as a rule, and in his *Atlas* (pl. xxiii. xxiv.) he differentiates between a pustular eruption, which is non-contagious and often associated with pediculi in the head, and his vesico-pustular impetigo contagiosa. The former frequently complicates the latter, he said, and so gave rise to confusion. On the other hand, J. Hutchinson, who was familiar with Startin's teaching, figured (*Syd. Soc. Atlas of Skin Diseases*, pl. xv. xx.) *common contagiosa porrigo* almost universally, but "probably accidentally," associated with pediculi capitis, and characterised by the formation of vesications, which dry into thick, dirty pus scabs. Extant drawings of a similar eruption secondary to vaccination crusts are alluded to. He thought the eruption produceable on an irritable skin by any kind of local irritation, and thence inoculable on other parts and on other persons by scratching. Coming to later times, Radcliffe Crocker holds that no line can be drawn between the epidemic febrile form of Tilbury Fox and the common non-febrile, more localised, cases of less definite course, which may have their origin in the effects of scratching, and are always due to the inoculation of contagious *pus* independently of its source. F. J. Payne characterises the lesions as vesico-pustules, and states that the original source of infection is always some kind of *suppuration*, such as the pus formation in pediculosis capitis, old vaccination scabs, conjunctivitis, otorrhœa, purulent rhinitis, ulcerative stomatitis, vaginitis, festered wounds or scratches; and many of these in turn may be secondary to impetigo.

We thus see that *impetigo* is the name given by most authors to an eruption of superficial vesicles, vesico-pustules, or pustules, arising from the inoculation of corresponding contagious exudations, and that for the most part the vesicular eruption described by Tilbury Fox is not distinguished as a separate affection from the primarily pustular phases, although his name *impetigo* (*porrigo*) *contagiosa* is widely adapted to include all. Duhring, however, distinguishes a primarily pustular impetigo. It must be clearly understood that in addition to the primary cases originating in the sources detailed by Payne, others in succession may be derived by direct or indirect inoculation,[1] and appear to be spontaneous, *i.e.* their source cannot be identified. Further, that similar eruptions, generally pustular, but differing as a rule in their wider dissemination, may appear as complications of a host of pruritic eruptions.

The all-important *bacteriological aspect* has next to be considered. It has been established that for the most part the staphylococci are the causal agents of pus. Other organisms, however, are also pyogenic, such as the bacilli of typhoid fever, glanders, tuberculosis, soft chancre, actinomycosis, and some ringworm fungi of animal origin. In 1887 an important paper by Bockhart appeared (*Monatsh. f. prakt. Derm.*), in which he proved that a form of impetigo with primary pustules, generally found about the nates and extremities, and often secondary to pruritic eruptions, was caused by staphylococci. He regarded furuncle and sycosis as different forms of the same disease. Since then it has been generally taught that impetigo, in the wide sense, is a staphylococcia. Soon observers were forthcoming who affirmed the streptococcic origin of some forms, and now the battle rages over this difficult problem. The latest author, Sabouraud, contributes (*Ann. de Derm. et de Syph.*, 1900) an elaborate memoir, in which he insists on the utter confusion which has arisen from not recognising a distinction between the streptococcic vesicular or phlyctenular impetigo contagiosa of Tilbury Fox and the staphylococcic pustular peripilar impetigo of Bockhart. This may be so, but putting on one side the latter eruption, many of us are still puzzled to know if we rightly include with the apparently spontaneous vesicular forms the more or less pustular phases arising from the various local sources detailed above. We may here note that the organisms in dispute are constantly present in the skin, and Payne supposes that "they in the first place cause inflammation, if the tissues in which they live are injured in any way; then growing as a pure cultivation in the inflamed tissue they acquire increased pathogenetic properties, and, by passing through one focus of inflammation after another, ultimately acquire sufficient virulence to cause suppuration in healthy skin." He notes that pus from deep abscesses does not appear to produce impetigo, and suppurative acne rarely.

IMPETIGO CONTAGIOSA OF TILBURY FOX (VESICULAR OR PHLYCTENULAR IMPETIGO) is a very common eruption, occurring especially in children and in the poorer classes, but occasionally seen in adults. It is auto- and hetero-inoculable directly and indirectly, sometimes quasi-epidemic, and varies from time to time in frequency and virulence. Sabouraud affirms that this eruption is due to a streptococcus, and the same which causes erysipelas, though Unna and Schwenter think it distinct. He points out that, if an ordinary blister be raised on the skin, hardly has the vesication risen before it is invaded by micro-organisms, and so it is with the impetigo vesicle. The streptococci are scanty and difficult to cultivate in the early stages, and are rapidly mixed with a secondary staphylococcic infection.[1] Now the pure streptococci impetigo is characterised by the evolution of tiny, *isolated*, inflammatory papules, which rapidly vesicate, or of clear vesicles, of a uniform type, without relation to a hair follicle and unaccompanied by any marked local disturbance of sensation. The vesicle remains clear as long as the causal streptococci culture continues pure, but becomes opalescent and pustular by leucocytic invasion consecutive to secondary staphylococcic infection. Secondary inoculations may contain mixed organisms from the first and furnish primary vesico-pustules, and, if the secondary staphylococci invasion be strong, even true pustules. The vesicles or vesico-pustules tend to enlarge into flat phlyctenæ, attaining in five or six days the size of a sixpence or shilling, unless ruptured by scratching, which generally occurs promptly. Even then a characteristic extending vesication can be often seen. The phlyctenæ are rarely full and pemphigoid (see Tilbury Fox's *Atlas*), sometimes simulate a vaccine vesicle, but generally are flat and collapsed. As in all excentrically spreading eruptions ringed or marginate lesions are not infrequently formed, and in rare case the prevalence of this type and their confluence produce strikingly figured pictures. The contents of the phlyctenæ dry up into crusts, often with a stuck-on appearance. If the contents are serous the crusts will be amber-coloured, but if puriform more or less thick and deeply coloured. The isolated lesions are often scanty, sometimes copious, and the crusts may be crowded in parts so as to form confluent patches simulating those of pustular eczema. The fall of the crust leaves a temporary red macule, which disappears without scarring. The initial evolution is generally local and on some exposed part, especially the face, or in the neighbourhood of the original source of the contagious exudation. Thence it may be inoculated on any other part, particularly the fingers, and in rare cases the greater part of the surface may be involved, and pemphigus be simulated. The mucous membrane of eye and nostrils may be implicated. On the limbs the lesions tend to assume the more formidable aspect known as *ecthyma*, and on the hands the vesications may attain a large size owing to the thicker corneous layer. I have seen a bag of serum hanging from a finger. Perionychia is not uncommon. True staphylococcic inoculations, such as we are about to describe, may also occur as complications, *e.g.* suppurative folliculites and even furuncles. The related glands may swell, but rarely suppurate. Tilbury Fox and others described an invasive febrile period, and a natural short and definite course, generally masked by the auto-inoculation of fresh lesions. The affection, however, is to be regarded as primarily a local one. I have twice seen a scarlatiniform rash arise from secondary intoxication.

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[1] *E.g.* by towels, pillows, washing flannels, antimacassars, football jerseys, etc.

It is probable that the so-called *pemphigus neonatorum*, sometimes epidemic in lying-in institutions, and a *contagious* "*pemphigus*" observed by Manson and others in tropical regions, belong here. Such infections own obvious sources, or may be apparently spontaneous, and they complicate a host of pruritic eruptions, and must be carefully distinguished from the original trouble. The ordinary localised phases of impetigo contagiosa are distinguished from eczema by the successive evolution of isolated lesions, without marked sensory disturbance, their tendency to peripheral extension and the formation of amber crusts of the discharge remain serous. Confluent areas present a real difficulty, but the presence of typical isolated lesions around generally affords a clue.

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[1] We must refer the reader to Sabouraud's memoir for a description of his special methods of cultivation.

The IMPETIGO OF BOCKHART is described by Sabouraud as of frequent occurrence and as an entirely distinct morbid entity due to the staphylococcus aureus, which, unlike the streptococcus, remains uncontaminated by other organisms. The typical lesion is a superficial pustule *ab initio* of greenish-yellow colour, centred by a hair, and surrounded, especially the younger ones, by a red areola. Such pustules may be of all sizes up to a pea, or even the end of the little finger, and they may be disseminated or crowded over the whole or part of a region, especially hairy regions, such as the scalp of the child, the moustache and beard of man. They may, however, occur anywhere. The pustules terminate in crusts, and when the latter fall the orifice of the follicle appears red and inflamed, and a deeper-seated folliculitis or a furuncle may result. Inoculation about the nail may set up paronychia and onychia. Striking characters are the frequent returns of the eruption, its sudden evolution in some hours, and the premonitory pain in the related lymphatic glands. This eruption can arise without obvious cause, or be secondary to all sorts of traumatisms, such as medicinal plasters, or a wet dressing, or such as happen to washerwomen, masons, and printers from various chemical substances. It can also complicate pre-existing affections, such as acne, the impetigo of Tilbury Fox, eczema (E. impetiginodes), prurigo, lupus, etc. This impetigo is auto- and hetero-inoculable, but there does not seem to be the tendency to excentric spread seen in the streptococcic impetigo.

Diagnosis.—The fundamental distinction between the staphylococcic pustule developed about a hair follicle, and the streptococcic phlyctena, without special relation to the hair follicle, which becomes complicated by staphylococci, and hence leads to mixed infections, has been sufficiently pointed out.

Treatment.—The recommendation of a multitude of remedies usually signifies difficulty of cure, but not in the present instance. The principles are to cleanse away, and, as long as new formations arise, to continue to remove, all infective vesicles or pustules, discharges or crusts. The latter should be softened by boric acid fomentations, carbolised vaseline, carbolic oil, and so on, and then thoroughly bathed away as fast as they form. All vesicles and pustules should be ruptured, and the infective exudations disinfected and removed by antiseptic washes made with boric or carbolic acid, or the biniodide or bichloride of mercury, and such like. The raw, exuding areas may be dried up by parasiticide and astringent lotions, or by mulls or plasters, or better still by ointments or pastes of oxide of zinc, to which 2 or 3 per cent of some parasiticide or antiseptic has been added, such as ammoniated mercury, naphthol, sulphur, carbolic acid, or salicylic acid. The following formulæ will serve as illustrations:—Sulphate of zinc 7 grammes, sulphate of copper 2 grammes, distilled water saturated with camphor and filtered 600 grammes. Make a lotion. Use to arrest early lesions (Sabouraud). Plumbi acetatis 1·0, acidi salicylici 2·0, zinci oxidi 20·0, adipis 50·0, petrolate 50·0. Infr. ung. (Dubreuilh). Acidi carbolici gr. v., acidi salicylici gr. x., pasta zinci oxidi ℥j. M. ft. pasta (Duhring). Hydrarg. ammon. gr. xij., ung. zinci benz. ad ℥j. M. ft. ung. (Tilbury Fox). Original sources of infection, such as purulent inflammation from mucous membranes, pediculi capitis, suppurating vaccination lesions, etc., must be sought for, and if they exist, cured. Lastly, not only must direct sources of inoculation be provided against, such as scratching, but indirect, such as soiled towels, pillows, chair and sofa coverings, caps, football jerseys, razors, and so on.

In the rarer, more or less universal cases, especially those sometimes seen in babies, the prognosis may be grave, unless the skin is thoroughly disinfected by prolonged antiseptic baths. The case has to be treated like a bad burn.

IMPETIGO HERPETIFORME

Under the titles *Impetigo herpetiforme* (Hebra), *Dermatite pustuleuse circinée et excentrique* (Besnier and Doyon), *Infection purulente tégumentaire* (Hallopeau), a rare malady of much gravity has been described, characterised by the formation of superficial pustules, tending to arise in patches spreading excentrically, which invades a great portion of the skin and evolves incessantly by successive outbursts accompanied by fever. At first it was supposed to occur exclusively in gravid females, like *herpes gestationis*, but in later years it has been described in women apart from pregnancy and also in man. The eruption has many analogies with the pustular phases of dermatitis herpetiformis, but in the latter the pustules are mostly formed, often very rapidly, out of vesicles; in I. herpetiforme the pustules evolve as such. Whether the eruption is due to a pyæmia, or toxæmia, or a neurosis, is not certain. Kaposi has published some striking portraits (*Archiv für Dermat.* 1887).

Impotence. *See* STERILITY.

Incontinence. *See* URINE.

Indicanuria. *See* URINE.

Indigestion.

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See also DIGESTION AND METABOLISM, and STOMACH.

THE term indigestion serves as a cloak to cover a multitude of dyspeptic disorders, some of which come throughout their entire course under the name, others only so long as clear evidence of the presence of a distinct morbid lesion is wanting, constituting the primary cause, and worthy of consideration in the light of an individual entity. Indigestion, in truth, applies simply to symptoms of digestive unrest of objective or subjective character, denoting presence of a morbid condition of the tissues and cells in some part of the alimentary system itself, or in other systems of the body, and reflexly influencing digestive organs and processes through the mediation of the nervous system or the circulation.

Thus patients suffering from symptoms common to early stages of gastric ulcer, cancer of the stomach, or dilatation of that organ, from the results that neurasthenia often occasions upon the processes of peptic digestion, those of a gouty habit may give rise to, or that may spring from changes in the composition of the blood and alterations in its supply, are as truly the victims of indigestion as those affected by simple gastric catarrh. Unlike the majority of diseased types indigestion cannot, therefore, be associated with any one principal causal agent. Nor can the individual symptoms even be considered as indicative of one or other separate pathological condition. Functional faults and organic error are so closely and intricately connected in result, and so surely exercise some influence on each other, that where the one is the other generally exists also.

Classification

- I. Acute.
  - A. From Organic Lesions.
    - 1. Acute Gastritis.
    - 2. Gastric Ulcer (occasionally).
    - 3. Gastric Cancer „
  - B. Through Nervous Influences.
    - 1. Cerebral.
    - 2. Spinal Gastric Crises.
    - 3. Peripheral.
  - C. Toxæmic.
    - 1. Autogenetic.
      - (*a*) Uræmia, Cholæmia.
      - (*b*) Auto-toxæmia.
    - 2. Heterogenetic.
      - (*a*) Alcohol.
      - (*b*) Chemical Drugs.
- II. Subacute and Chronic.
  - A. From Organic Lesions.
    - 1. Simple or Chronic Gastritis.
    - 2. Gastric Ulcer (occasionally).
    - 3. Gastric Cancer.
    - 4. Dilatation of Stomach from Pyloric Stenosis.
    - 5. Gastric Cirrhosis.
    - 6. Tubercle.
    - 7. Syphilis.
    - 8. Venous Congestion.
  - B. Functional.
    - (I.) Of Local Origin.
      - 1. Secretory.
        - (*a*) Acid.
          - (1) Hyperchlorhydria.
          - (2) Gastroxynsis.
          - (3) Hypochlorhydria.
        - (*b*) Ferment.
          - (1) Hypopepsia.
          - (2) Apepsia.
      - 2. Sensory.
        - (*a*) Sensation.
          - (1) Heartburn.
          - (2) Pain after or before food; Gastralgia.
        - (*b*) Sensibility.
          - (1) Tenderness on pressure.
          - (2) To peristaltic movements.
      - 3. Motor.
        - (*a*) Hyperperistalsis.
        - (*b*) Hypoperistalsis.
        - (*c*) Pyloric Insufficiency.
        - (*d*) Pyloric Spasm.
        - (*e*) Dilated Stomach.
        - (*f*) Gastroptosis.
        - (*g*) Delayed emptying.
    - (II.) Of Distant Origin.
      - 1. Cerebral.
        - (*a*) Hysteria, Neurasthenia.
        - (*b*) Hemicrania.
      - 2. Spinal.
        - (*a*) Spinal Irritation.
        - (*b*) Some cases of Systemic Disease.
      - 3. Peripheral.
        - (*a*) Intestinal Lesions.
        - (*b*) Pelvic Disease.
        - (*c*) Pregnancy.
        - (*d*) Enteroptosis.
  - C. Toxæmia.
    - 1. Autogenetic.
      - (*a*) Uræmia, Cholæmia, etc.
      - (*b*) Auto-intoxication.
      - (*c*) Gout and Rheumatism.
    - 2. Heterogenetic.
      - (*a*) Alcohol.
      - (*b*) Drugs and Poisons.

The classification of cases of indigestion introduced above must not be regarded as being in any way either exhaustive or complete. Space forbids a further elaboration. As it stands it is meant to indicate the mode of origin and the nature of the primary cause of the various chief types of indigestion, apart from the many forms in which more than one of these factors coexist, and from those in which different symptoms are produced by similar causes. Every case of indigestion of organic source must necessarily exhibit some disturbance of function.

Those disorders of digestion which are purely functional from the first may continue without establishment of any organic change for some length of time, but if very prolonged generally induce actual changes in the tissues and cells of the stomach walls.

It cannot be too strongly insisted on that indigestion must on every occasion be regarded merely as a symptom, not as a disease to be treated by rule of thumb with routine remedies and stereotyped therapeutic measures. Few cases are identical in form, origin, or personal idiosyncrasy. In many the primary source from which the symptoms proceed has no direct relationship to the stomach or its digestive processes,—the morbid symptoms, subjective or objective, exhibited by that organ depending entirely upon irritation of the nervous tissues supplied to more or less distant parts, and consequent abnormal stimulation reflexly conveyed to the gastric nervous mechanism.

Again, constitutional affections may be really answerable for the occurrence of the symptoms, and unless they are dealt with along with the treatment of the local complaint, may inhibit success. In anæmia, gout, chronic debilitating diseases, little benefit will accrue unless the larger lesion is recognised as the more important factor in the management of the less.

Another class of case confesses to toxæmia as the *causa causans;* autogenetic when produced by excessive absorption of poisonous products of intestinal fermentation, proteolysis, or putrefaction, or by inability on the part of the liver to render the quantities conveyed to it innocuous; when aroused by presence of the results of metabolism in the blood-stream, because of renal inefficiency, or of biliary items where their passage into the bowel is arrested; heterogenetic when induced by alcoholic excess, or by ingestion or introduction of various gastric irritants into the stomach or body.

The general neuroses also must be borne in mind,—hysteria, neurasthenia, and mental depression,—in the consideration of indigestion; and in women, the wonderful influence exercised by diseased conditions of the organs of generation upon digestive processes, especially of the ovaries, as well as the intimate connection which the menstrual functions and pregnancy bear to them.

Still another point worthy of attention relates to the tendency that exists in cases in which certain glandular secretions, for whose real significance in the bodily economy we are still groping, have been arrested, or altered,—as, for instance, the secretion of the thyroid gland in myxœdema and goitre, or of the ovaries after double ovariotomy,—to the occurrence of dyspeptic troubles. Recognition of the fault underlying such cases will elucidate many of their puzzling symptoms, and indicate the most advantageous line of treatment.

Further, as "digestion" signifies all the processes by means of which the nutritive elements of the food introduced into the body are adapted for absorption into the circulating fluids for use, for assimilation, and in metabolism, so "indigestion" may be held to include many more recondite morbidities connected with the retail department of co-operative protoplasmic commerce, than the wholesale division, dealing with crude material, can offer. From the time food is taken, up to the evacuation of that which is waste, and until the nutritious materials separated from it have been finally transformed into cell components, or paid their way in the production of energy at the cost of their own decomposition, digestion continues and indigestion is possible.

Robin's classification divides dyspepsias into three groups: dyspepsias from exaggeration of function, or hypersthenic; those due to insufficient function, or hyposthenic; and those caused by perversion of function, or gastric fermentations. Einhorn recognises organic disease with constant lesions, functional disease with variable lesions, and nervous affections of the stomach.

Etiology.—Indigestion respects neither age nor sex; male and female, old and young are liable to be attacked. Both age and sex, however, have some influence over the incidence of the malady, and the type.

Predisposing Causes.—1. *Dietetic.*—Undoubtedly the chief source of indigestion lies in errors of diet. It is a very difficult and hazardous matter to define what constitutes a dietetic error in the universal sense. One man's meat is another's poison. Use and wont within certain limits influences the question. Habits, occupation, temperament, constitution, and bodily condition modify the conception of what may be appropriate in each separate case.

Idiosyncrasy also has to be remembered.

Mere personal errors, apart from these considerations, consist in habitual omission to properly masticate the food, in hurried bolting of meals, immoderate use of alcoholic stimulants, of astringent infusions, ice-cold liquids, or over-hot drinks. In addition, where the circumstances of the patient place no restriction in the way of his or her indulgence of appetite, the continual enjoyment of highly spiced foods, the ingestion of one of the three primary forms of food-stuffs out of all proportion to the other two, whether it be proteid, carbohydrate, or fat, and a too close sequence of heavy meals, tend in time to produce dyspeptic symptoms, either from local quarrels on the part of the digestive organs, surfeited by excess, or from general conditions, reflexly causing a disordered state of the alimentary tract.

Indigestion from long-continued dietetic error, whether it be due to personal misdemeanours, to constitutional states, or to environment, chiefly arises from incongruity between the food taken and the physical needs of the body;—the amount of active expenditure of muscular energy, by inclination or of necessity, corresponding to but part of the nutriment ingested and absorbed;—or from unsuitable characters of the food for the upkeep of function—mental, secretory, or trophic. In those to whom circumstances deny a choice, irregular, unsuitable, and insufficient forms of sustenance, badly prepared or not prepared at all, often initiate chronic indigestion.

2. *Habits.*—Any ill effects which may follow such dietetic errors as are enumerated above are dependent in great part on the habits of the individual. Indiscretions in diet, productive of serious disturbance in the digestive economy of the sedentary occupant of an office, or of one largely confined by necessity to the house, may prove harmless to those who live an outdoor life, or whose occupation entails a considerable output of physical energy. But conversely, a monotonous or barely sufficient diet may serve for one who expends but little muscular force, while quite inadequate for the worker and a source in him of digestive disorder. A very common starting-point for indigestion is shown by persons, accustomed previously to an outdoor life or much open-air exercise, who take up their abode in towns, or enter upon confined or sedentary occupations. The dietetic habits formerly suited to their circumstances are too often continued under these changed conditions, and if long maintained almost certainly result in ill-health.

SYMPTOMS.—Owing to the fact that many of the clinical signs and symptoms of indigestion are common to more than one of its different types, perhaps the most simple and intelligible course to follow is to discuss them *seriatim*, as they fall under (1) Sensory, (2) Motor, and (3) Functional groups.

1. *Sensory Symptoms common in Indigestion.*—Local sensations of discomfort in the stomach are generally the first signs given of the presence of gastric disorder. They may only amount to a vague feeling of uneasiness, of a ball in, or some apparent weight pressing down, the stomach; again, they may take the form of a gnawing pain, persisting for some time, and usually directly related to the ingestion of food, or of more paroxysmal attacks of pain, either at some special stage of gastric digestion, or independent of digestive processes. Not infrequently the subjective sensation of pain is more or less definitely confined to one point, and can be located by the patient. In the majority of cases, however, the feeling of uneasiness and pain is diffuse and referred to the entire organ. Another common complaint is that of "swelling" of the stomach, a feeling as if it were greatly distended, and had too little accommodation for its bulk. This may arise from actual distension of the stomach with gas, or occur apart from any real dilatation or obstruction, and due to a hyperæsthetic condition of the sensory nerve fibrils in the wall. Patients now and then describe their stomach as giving them the idea of an absolutely inert mass or foreign body,—a symptom of nervous dyspepsia with temporary arrest of peristalsis and secretion, where the nerves of the surrounding abdominal viscera are hypersensitive. Sensations of dragging down of the stomach are often very distressing; they appear in dilatation, in gastroptosis, where the pyloric end alone is displaced downwards—pyloroptosis,—frequently with floating kidney, and where peritonitic adhesions exist between the stomach and other abdominal organs or tissues.

The relationship between gastric uneasiness and painful sensations and the taking of food is an important factor in cases of indigestion. These symptoms may precede, accompany, or follow its ingestion. If coming after food the length of time which elapses before the morbid sensations set in is of value in determining their origin. When distress and pain occur at the time of entry of food into the stomach, if they be not more than slight though persistent, subacute or chronic catarrh of the gastric mucous membrane, malignant disease, dilatation with incomplete emptying and fermenting residual contents, or irritability of the nerve-endings in the mucosa, resenting the introduction of new material, may be present. More acute pain and discomfort coincident with entrance of food occur in gastric ulcer, in cases of cancer with ulceration, and in hyperæsthetic nervous cases.

The usual site of pain due to gastric disease is the epigastric region, extending up below the sternum in chronic gastritis, of a radiating and more continuous character in cancer of the pylorus, severe and strictly circumscribed in gastric ulcer, but associated with a similar small painful area in the back close to and usually a little to the left of the eleventh or twelfth dorsal vertebral spine; paroxysmal and localised in idiopathic gastralgia; dull and more diffuse in neurasthenia. Cancer of the smaller curvature of the stomach may give rise to painful sensations at a somewhat higher level, of the posterior wall to pain felt in the back. In dilatation and gastroptosis the location of the pain approaches nearer to the umbilicus. Gastralgia when caused reflexly by disease of other organs is usually located in the epigastric region, but the pain tends to spread over a wide area.

Cardialgia, a symptom commonly described as heartburn, proceeds from irritation of the nerve terminations in the lining membrane of the œsophagus and pharynx by hyperacid contents of the stomach, eructated by antiperistaltic action and initiated by over-stimulation of the gastric nerves by the excessive acidity of the contents. Heartburn is merely an extension of the less definite variety of pain induced by excess of acidity in the stomach itself to the œsophagus and pharynx, whose nerve-endings are more sensitive to acid stimuli.

2. *Motor Symptoms.*—The most frequent result of disturbance of gastric motility is delayed digestion, from weakened peristaltic movements of the muscular coat of the stomach wall, with some degree of stasis of the contents and fermentation. If severe or protracted, actual dilatation may follow. Hyperperistalsis is not so common, but is met with in irritable nervous cases, especially in those with hyperchlorhydria, and occasionally is so pronounced in character that the contractions are palpable. Pyloric spasmodic contractions are of a similar nature; irritation of the adjacent gastric surface by acid contents inducing the paroxysms which are frequently accompanied by severe cramp-like pains and burning sensations.

Vomiting or emesis is directly due to abnormal motor action, but is usually a secondary result of nerve irritation. Consisting in paroxysmal gastric antiperistalsis, combined with contraction of the diaphragmatic muscle and of the muscles of the abdominal parietes, it may be of local, remote, or central origin. If of local origin, the exciting cause may be in the stomach contents, or in the blood supplying the local nerve ganglia; if remote, the primary source is usually irritation of peripheral nerve fibres or cells, of the abdomen and pelvis in pregnancy and ovarian tumours, of the peritoneum in acute peritonitis and intestinal obstruction; and, if central, either of spinal or cerebral origin, from organic lesion, toxæmic irritation, or functional nervous disorder. Generally speaking, acts of vomiting which are independent of meals, are preceded by little nausea, and followed by few if any feelings of ill-health, and in which the materials vomited are of normal character, or nearly so, point to a functional cause, peripheral or central, uncomplicated by local organic disease, or by morbid changes of local function induced by neurotic influence. The relationship between the taking of food, the kind of food, the regularity or irregularity of occurrence, the degree of nausea experienced, the character and amount of the vomit, the features of the act itself, and an attack of emesis, requires investigation before the true significance of the symptom can be properly appreciated; in addition, of course, to the obtainal of positive or negative evidence as to the presence of complicating lesions, which may possibly be to blame.

Rumination or merycism is another motor symptom, and one indeed which is not so uncommon as popularly supposed. It may be, however, perfectly compatible with a healthy state of digestion, although tending to initiate symptoms of dyspeptic disturbance in course of time, if the habit be long and constantly persisted in. While met with in certain cases of indigestion, particularly in those of neurotic origin, it can scarcely be regarded as a true morbid symptom of dyspepsia, but rather as a more or less abnormal habit, often begun in curiosity, perceived to be not unpleasant, repeated too frequently, and ultimately found to have become a necessary custom. The habit has arisen from imitation, as in Körner's case, and has been traced in a few instances to hereditary influence.

3. *Functional Signs and Symptoms.*—These denote the numerous errors of secretory and digestive power. The methods of examining the contents of stomach and the significance which attaches to their results have already been fully discussed in an earlier article. It will suffice here to introduce a scheme of classification indicative of the relationship between altered characters of the stomach contents and indigestion in many of its forms:—

CLASSIFICATION OF GASTRIC CONDITIONS WITH REGARD TO THE CHARACTERS OF THE STOMACH CONTENTS, CHEMICAL AND PHYSICAL

I. Acidity.
  A. Normal.—In health; hepatic dyspepsia; cancer of cardiac orifice and œsophagus; erosions; nervous and anæmic dyspepsia.
  B. Hyperacidity.—1. Hydrochloric acid increased.
    In gastric ulcer, and erosions; from nervous causes; in some cases of dilated stomach; occasionally in cancer following ulcer.
    2. Hydrochloric acid diminished; organic acids increased.
    In cancer of the pylorus and pyloric region; dilatation of the stomach; nervous dyspepsia; the later stages of gastric ulcer; chronic catarrh; atonic dyspepsia.
  C. Hyperacidity.—1. Without organic acid fermentation.
    Hypochlorhydria without fermentation; at beginning of acute gastritis, or during convalescence from it; cancer of the stomach (rarely); cirrhosis ventriculi; during acute or chronic febrile and cachectic disorders (on occasion).
    2. With organic acid fermentation.
    Chronic catarrh; atrophy of the mucous membrane; cancer; waxy degeneration; cirrhosis; acute febrile diseases; dilated stomach without pronounced stasis; phlegmonous gastritis.
II. Digestive power.
  A. Normal.—In health; gastric ulcer; nervous dyspepsia; many cases of œsophageal and cardiac cancer; early catarrh.
  B. Increased.—Gastric ulcer; nervous hyperchlorhydria; often in diabetes.
  C. Diminished.—Cancer of stomach; chronic catarrh; atony; dilated stomach with catarrh; nervous dyspepsia; acute gastritis; acute fevers; cirrhosis.
III Length of stay in stomach.
  A. Normal.—In health; gastroptosis without obstruction; nervous dyspepsia; gastric ulcer.
  B. Shortened.—Hyperchlorhydria; hyperperistalsis; insufficiency of pylorus; gastric ulcer (rarely), diabetes.

C. Lengthened. — 1. Diminished peristalsis. Nervous dyspepsia; cancer; dilatation; pyloric stenosis; cicatrices of old ulcers.
  2. Decreased power of digestion.—Cancer; chronic and atrophic catarrh; acute gastritis.
  3. From both causes.—Cirrhosis of stomach; dilated stomach with catarrh; many nervous dyspepsias; hypersecretion (fluid remaining in form of gastric juice, not of food). Cancer at later stages.

IV. Specific gravity (after filtration).—1. Normal. After a meal 1010-1020.
  2. Increased. Hyperacidity 1020+
  3. Diminished. Hyperacidity 1010−

V. Blood.—1. Bright red, copious, coagulable. Gastric ulcer.
  2. Darker, copious, clotted. Gastric ulcer or cancer; duodenal ulcer (rarely).
  3. Bright red or brown, smaller amount.
  Gastric erosions, chronic ulcer, with weak cicatrix; acute gastritis; hepatic congestion; venous engorgement.
  4. "Coffee grounds." Cancer, especially with atony and dilatation.

SPECIAL FORMS OF INDIGESTION

Hemicrania (Migraine, Megrim, Sick-headache).—The subject of megrim, its nature and significance, to be adequately discussed, would require a volume to itself. The question whether hemicranial attacks are species of nerve-tornadoes, as advanced by Liveing, are auto-toxæmic in origin, are caused by both of these factors in combination, or derived from various other suggested sources, boasts of so extensive a literature and has been so diversely answered that it is hopeless to go fully into it here. To the writer the most probable theory, and the one capable of the widest application, ascribes the exciting cause to a sudden overflow of abnormal nerve force, similar to that which occasions epileptic seizures, but expended upon sensory and functional nervous mechanisms in lieu of motor areas, incited thereto by the presence of poisonous substances in the blood, the products of insufficient metabolism, which, when they attain to a certain proportion in the blood, initiate a critical paroxysm, followed by a longer or shorter period of quiescence. Megrim, associated with gouty habit, is explicable on similar grounds. When intimately connected in women with the menstrual function, the periodical stimulus to the nervous system thus afforded suffices to liberate any morbid surplus of nerve energy, in conjunction with the usual concomitants of such cases,—constipation and some degree of auto-intoxication. Long made a scapegoat in megrim, the liver is more probably a companion in misfortune than an organ meriting blame.

The symptoms accompanying attacks of megrim are most variable in character; practically the only features which are constantly presented being the paroxysmal nature of occurrence, the entire absence of cognate symptoms throughout the intervals, and the intense feeling of nausea produced during the later stages of the attack. Hemicrania; ocular disturbances, such as hemianopsia, scotoma, and subjective perception of apparent visual objects, coloured or uncoloured; mental confusion, paræsthetic symptoms on one side of the body, and post-paroxysmal nervous phenomena,—aphasia, temporary loss of memory,—may severally be present or absent.

Vomiting, or some other form of critical evacuation, usually intervenes before subsidence of an attack; but this is not invariably the case, the nausea sometimes disappearing gradually apart from actual crisis.

Flatulent Dyspepsia.—Gastric flatulence may be of two kinds: either the result of fermentative processes taking place in the contents of the stomach, or of nervous origin. When fermentation of food-stuffs constitutes the source of the gas there usually coexists a lack of hydrochloric acid, secretion of which has diminished owing to catarrh of the gastric mucous membrane, atrophy of its cells, or nervous inhibitory action; and the tendency towards gaseous evolution is increased in each instance if some degree of stagnation of the gastric contents is also present. Flatulence of this nature corresponds in degree and in character with the variety of food ingested; the more readily fermentescible the food-stuff the greater the ebullition of gas. Should the stomach be dilated, and residual remnants from former meals be left in it after the normal time of emptying, while the proportion of hydrochloric present is insufficient to prevent fermentation, the symptoms of flatulence may obtain irrespective of meal-time or form of food. The gas proceeding from fermentative processes generally possesses an odour corresponding to the nature of the process producing it; is most abundant after the ingestion of farinaceous foods; and is increased by the administration of alkalies. In rare instances the presence of marsh gas has been detected by its ignition on contact with a flame of the mouth.

In many examples of purely nervous dyspepsia, unassociated with fermentative processes, symptoms of flatulence are often met with, astonishing in the volume of gas evolved. Here the formation of gas is independent of the character and the ingestion of food; the gas itself is odourless, tasteless, is apparently expelled without effort or much discomfort, and may be due either to excessive entrance of carbonic acid gas from the blood stream into the stomach cavity, owing to abnormal nerve action, or to expulsion of atmospheric air previously swallowed. Indeed, air-swallowing and eructating forms a special type of neurotic indigestion.

Indigestion in Dilatation of the Stomach.—The degree of dilatation present modifies the character of the symptoms shown. In minor forms, where enlargement of the stomach scarcely amounts to true dilatation, the *megastria* of Ewald, characteristic symptoms may be absent, only a few of the signs common to chronic gastric catarrh of mild type attracting notice. Where the degree of dilatation is more pronounced, symptoms indicative of delayed emptying of the stomach-contents, of stasis, and fermentative processes in the residuum left in the viscus, appear in most instances. But it is of importance to remember that the symptoms presented by the possessors of a dilated stomach vary directly with the cause of the dilatation. Where the condition has arisen as the sequel of habitual over-distension of the organ with food or with fluid, leading ultimately to loss of muscular tone, the chief fault lies not so much in loss of digestive power as in defective motility; where dilatation follows atonic dyspepsia, after long-continued chronic catarrhal conditions have injured both the secretory and motor functions of the stomach, stasis of the contents is aggravated by deficient digestive properties; in the by no means infrequent examples of dilatation, to all appearance solely due to enfeebled resilience and motility through lack of efficient nervous control, a most incongruous series of symptoms may be presented, the ordinary symptoms of a dilated stomach being masked by some of the many possible in nervous dyspepsias; and, lastly, where dilatation of the stomach depends upon a stenosis of the pyloric orifice, the symptoms and signs correspond. In addition, it should be further mentioned that the signs accompanying gastric dilatation from pyloric stenosis vary with the nature of the constriction. Omitting consideration of cancer of the pylorus at present, simple hypertrophy of the pyloric muscular and connective tissue coats may lead to narrowing of its lumen, and prove a hindrance to the escape of stomach contents, and contracture of cicatricial tissue over the site of an old ulcer acts similarly. Occasionally hyperacidity of the gastric contents leads to temporary spasmodic closure of the pyloric sphincter by reason of irritation of the nerve-endings in the gastric mucous membrane; and, if frequently repeated, dilatation of the stomach may be a result, more especially where persistent irritation and consequent contracture has induced an actual hypertrophy of the pyloric tissue.

Analyses of the gastric contents in cases belonging to these several classes generally yield characteristic points of difference. Wherever stasis accompanied by loss of peptic power and deficiency of hydrochloric acid has taken place, examination of the contents affords evidence of an excess of organic acids of fermentative processes, and of decreased digestive activity. Should the contents be obtained from a dilated stomach of nervous origin, or following upon cicatricial pyloric obstruction, they may prove to contain hydrochloric acid in normal or excessive proportion.

Associated with Displacement of Abdominal Viscera.—This variety of indigestion is fully treated of elsewhere (Gastroptosis, Enteroptosis, Floating Kidney), and need not be further alluded to here, except to emphasise the possibility of dyspeptic symptoms arising from such lesions.

Œsophageal diverticula, congenital or acquired, may give rise to very pronounced symptoms of indigestion, attended with great difficulty in diagnosis. The special features shown by such cases chiefly consist in sudden acts of regurgitation of food, unassociated with nausea and the ordinary phenomena of emesis; the regurgitated material differing in composition from gastric contents, lacking hydrochloric acid and pepsin, although frequently of acid reaction from the presence of organic acids of fermentation, and as a rule including portions of food swallowed some time, some days even, beforehand.

Enough has already been said as to the factors which have a part in causing the appearance of dyspeptic symptoms, and whose origin must be sought for elsewhere, to indicate the necessity of careful investigation in all possible directions before a trustworthy diagnosis can be arrived at. This matter is so important that it may again be insisted on, that recognition of the probable existence of a primary cause, and appreciation of its true nature, are of more value in connection with indigestion than the greatest familiarity with the local symptoms common to it.

Treatment.—In few conditions is an intelligent and rational perception of the close relationship between cause and effect so valuable as in the treatment of indigestion. And, further, owing to the fact that the cognomen, indigestion, refers to symptoms of varied origin, although it may be similar in character, it is of the utmost importance to ascertain the nature of the true, as compared with the apparent, cause which produces the effect, before any satisfactory and efficient course of treatment is decided on.

Simple, passing attacks of mild indigestion, arising from brief and temporary errors of diet, of habit or circumstance, and characterised by the symptoms common to slight gastric catarrh, readily subside if a light diet be ordered, the exciting cause not repeated, and the bowels freely moved with one or other of the milder purgative drugs; if thought advisable, even if only as a "placebo," a gastric sedative may be given: one of the different forms in which bismuth is prepared, with or without alkaline carbonates. The main indication lies in stoppage of the direct source, light diet, never taken in large bulk at one time, and free evacuation of the bowel.

Acute cases of gastric catarrh, due to alcoholic excess, to severe local inflammation from irritating articles of food or toxins, or occurring during the course of subacute and chronic attacks, either from the principle of the summation of stimuli which in these cases accrue from long-continued dietetic irritation, or from ingestion of more than usually irritating material, benefit most from prompt emptying of the stomach, by giving an emetic, if indeed nature has not anticipated this, and from exhibition of a rapid purge, saline or cholagogue, to rid the intestine of toxic bodies. Milk and slops—no alcohol, tea, or coffee, no vegetables or fruits, cooked or raw—should form the diet, given every two or three hours during the day. Water, cold or hot,—aërated, with or without potash or soda, or mixed with the milk,—may be taken *ad libitum* as is found agreeable. The local gastric distress is best relieved by the application externally of heat or of a mustard plaster. The common coincidence of duodenal and acute gastric catarrh, leading to catarrhal jaundice, should be kept in mind, and the adoption of measures appropriate for the removal of this condition in addition to those directed against the gastritis will often prevent this sequela.

As few cases of mild indigestion of brief duration, or of acute gastritis, are due to more than a temporary inflammation of the gastric mucous membrane from irritating food-stuffs, or too large meals, the majority are best treated by regulation of diet and habit, and by giving the stomach a period of comparative rest; the less medicine used the better.

If, however, such attacks are accompaniments of more general morbid conditions, such as anæmia, chlorosis, gout, valvular heart disease, portal obstruction, and neurasthenia, it is not sufficient to treat them alone apart altogether from the really more important general lesion. In truth, under these circumstances it is more difficult to treat them successfully at the time, while they display an immensely greater tendency to recur.

But it is in the chronic and protracted subacute types of indigestion that the question, Which line of treatment is proper for adoption? comes to be hardest to answer—that the importance of recognising the nature of the primary source of the evil ranks highest. For here vicious circles have usually been established. Recurring attacks of simple or acute gastric catarrh have led to chronic inflammatory changes; deficient peptic digestion to intestinal disturbance; diminished absorption of nutriment to metabolic error, to anæmia, gout, inertia, and nervous debility; or *mutatis mutandis* anæmia, gout, reflex influences, and neurasthenia have induced dyspeptic symptoms. Whether the indigestion precede the other conditions, or *per contra* arise from them, their coexistence aggravates both forms of disease, and direct treatment of the one is often injurious to the other.

The problem then to be solved is whether it would be best to deal first with the dyspeptic condition or with the more general error. The answer depends largely upon the presence or absence of organic lesion in the gastric mucosa, upon whether the processes of digestion are actually amiss, or only subjectively annoying; whether the general or the local condition is the more important; and upon the greater likelihood of benefiting the one by preliminary attention to the other than *vice versa.*

Alkalies given before meals tend to increase the acidity of the gastric contents after them; dilute mineral acids, too, act conversely. Given with or after food they exactly reverse their actions. The effect, however, of alkalies given with or after food in lowering the acidity of the stomach contents is purely equational, and exactly corresponds in degree and duration to the amount introduced. If sufficient time elapses before emptying of the stomach the decrease may become increase, the presence of the alkali inciting an enhanced secretion of hydrochloric acid. As the secretion of this acid is governed by the force of the exciting stimulus, and displays an intimate connection with the degree and character of gastric acidity present, the introduction of alkalies into the stomach calls for greater secretion of acid, of acids or less.

Alkaline drugs, therefore, given to quell acidity in reality serve ultimately to heighten it; acids recommended for hypo-acidity are liable to weaken in place of strengthening the already deficient acid secretion. These statements only apply when disturbing complications are absent.

The therapeutic value of the digestive ferments is a disputed matter. Predigested food often proves of great if temporary service; but the exhibition of pepsin, pancreatin, etc., by the mouth most probably exerts less beneficial effect in gastric errors than has been ascribed to it by some.

Dilute hydrocyanic acid in small doses frequently repeated, with or without some form of bismuth, often suffices to sooth gastric pain; now and then, if severe in character, morphina or codeina may be required.

Instrumental Measures.—Lavage is undoubtedly of service in certain cases, but its beneficial properties have probably been much exaggerated, and certainly its practice has often been abused. Lavage may be imperative in pronounced dilatation of the stomach, or excessive pyloric stenosis; in other cases the influence of contemporary treatment, medicinal and dietetic, and the effect upon the psychical centres exerted by the details of the process, require to be eliminated. Where removal of the stomach contents from time to time is not obligatory by reason of some absolute hindrance to their downward passage, lavage if habitual is unnatural, illogical, betokening absence of resource on the part of the physician, or the induction of a habit contributed to by him.

Electricity applied to the surface of the stomach itself, externally over the gastric region, or over the segments of the spinal cord related to it, sometimes produces very beneficial results; but the effects produced by it are of so variable a nature, that actual trial alone can suffice to prove whether it may be of service or not in any particular case. Further allusion to the merits or demerits of these, and of the many other instrumental means of intragastric treatment which have been devised, goes beyond the scope of this article.

Diet.—It is as important to base the choice of a suitable diet upon the nature of each individual case, as it is proper to modify the lines of medicinal treatment upon the same. Too frequently some particular form of diet comes to be recommended as a routine practice for all dyspepsias, whatsoever be the type. The favourite diet may vary from time to time, seldom from case to case. Personally the writer has little faith in, and considerable distrust of, the imposition of strict, rigid rules for diet, of restriction within narrow limits as to food, definite, or what is still worse, indefinite in duration. In simple transient catarrh, or in cases of acute gastritis, brief limitation to the simplest food proves excellent. In the majority of the more prolonged cases of indigestion, and these are the cases which most often call for medical treatment, a longer or shorter course of restriction to a few specified food articles frequently takes the form of punishment, and defeats its own object by arousing feelings of repulsion through the constant sameness of the viands permitted, and of depression consequent thereon. Monotony deadens enjoyment, weakens appetite, injures digestive power, while increasing morbid nerve influence.

It were more rational to be guided by signs shown by nature, and to endeavour to facilitate the progress towards recovery along natural lines, than to institute for a considerable period of time immutable and cribbed conditions, disregarding any changes which may occur intercurrently—conditions as unlike the salutary variations always exhibited in nature's doings as is possible.

How far psychical changes in temperament represent concrete alterations of substance in nerve ganglia, or differences in the processes productive of nerve force, is as yet undecided; be the relation what it may, psychical temperament exerts marked influence over functional activity throughout the body, and concerns our methods of therapy. All that tends to depress the mind tends to diminish functional activity, and to intensify or prolong existing morbid conditions. The dietetic rule, which, while benefiting the more local errors of digestion, is best calculated to stimulate the psychical functions, and not to cause mental depression, which progresses in variety of available foods as the condition of the patient improves, while strictly remaining within the limits of tolerance, is the rule, the adoption of which is likeliest to be followed by satisfactory results, general as well as local, and lasting in effect.

Gastric processes are principally concerned in the digestion of proteids, in the disintegration of masses of flesh, and in the thorough intermixture of all the food-stuffs taken. Normally it has little or no action on carbohydrates or fats, except by reason of its acidity in preventing their fermentation. Wherever the secretion of hydrochloric acid is decreased, and, therefore, abnormal fermentative changes in carbohydrate substances facilitated, farinaceous foods should be largely forbidden, especially those which are most readily fermentescible. The diet given, varied according to circumstance, should be mainly composed of the blandest forms of proteid foods, never taken in large quantity at any one time. The normal powers of digestion possessed by the stomach juice are lessened; they should, therefore, be given an easier task to do, while guarded as far as possible from further irritation.

Indeed, it almost seems to be quite rational to assume that whenever the stomach is sick the diet should mainly consist of that class of foodstuffs which is naturally acted upon by its secretions, discarding the foods which are almost as foreign bodies to it.

Nervous Dyspepsia.—The multitudinous manifestations of indigestion proceeding from nervous causes require both local and general treatment. Here, again, the subject is complicated by the question of *post hoc, vel propter hoc.* Are the symptoms the result of morbid nervous affection, or have the dyspeptic lesions brought on nervous disorder? While, again, the possibility of a purely reflex causation of the symptoms of indigestion adds to the difficulty.

As a rule local treatment can only be palliative, although in so far as it benefits the general nutrition of the body it helps towards ultimate cure; while removal of the source of the nervous disturbance is the real goal to be aimed at. If the symptoms arise reflexly from local disease in other regions, as is frequently the case, relief will follow most surely from the cure or improvement of the remote causal agency. Diseased conditions of the large bowel and rectum, of the female pelvic organs, displacements of other abdominal viscera than the stomach, pregnancy, and disordered menstruation, may require attention. In several instances the writer has procured rapid relief of dyspeptic symptoms in women at or past the menopause, or who had undergone double ovariotomy, by the sole employment of ovarian extract or didymin—all local calls for treatment being entirely ignored. When the underlying cause consists in nervous debility, neurasthenia, or nervous irritability, choice of correct therapeutic measures is a matter for grave consideration. Treatment of symptoms in accordance with the nature of the local lesions inducing them, as revealed by analytical examinations of the gastric contents, or in the light of previous experience of like cases, prohibition of dietetic transgressions, and prescription of nerve sedatives may help. Far more benefit follows change of habit, of environment, and of interest; less (or more) professional work or manual labour; less (or more) physical and mental exercise; less (or more) opportunity for careful regard to personal traits, phenomena, and wants. Many of the victims of nervous dyspepsia, who have developed hypochondriacal tendencies, derive special benefit from change of scene and circumstance, with absence from routine work, and diversion of thought. Others, again, seem to experience a larger degree of relief from assiduous local therapy, especially if employment of physical methods be associated with it.

Excessive emaciation in neurasthenic subjects, with neurotic dyspepsia, especially that type known as *anorexia nervosa*, can only be successfully treated away from home and by some form of the treatment associated with the name of Weir-Mitchell, into which rest, massage, and a generous supply of simple food enter.

In Anæmia and Chlorosis.—In treating the symptoms of chronic indigestion so often met with in anæmic and chlorotic subjects the possibility of gastric ulcer should always be borne in mind,—acute, in course of formation, and existing in part, or chronic, imperfectly healed, or cicatrised. The increased sensitiveness of the nerve endings in the gastric mucous membrane towards the irritant effects of free acid in the contents, because of the lowered nutritive properties of the blood supplied to them, must also be taken into account. Their digestive powers may be normal, along with severe symptoms of gastric disorder, arising solely from the anæmic state of the blood.

When the symptoms observed point to irritative dyspepsia, and the gastric contents contain a normal proportion of free hydrochloric acid, or even a somewhat diminished amount, treatment should be directed towards mitigation of the irritant effects of the free acid and improvement of the state of the blood. In such cases many of the ferrous salts are well tolerated.

It is a much more troublesome matter to treat successfully anæmia complicated by atonic and hypo-acid indigestion, for hæmatinic drugs are ill borne by the stomach, while measures devised for removal of the gastric condition are often prone to harm rather than benefit the anæmia. The most rational method to adopt, perhaps, is to endeavour, in the first place, to improve the powers of gastric digestion, giving at the same time such non-irritating tonic drugs, not necessarily ferruginous, as strychnine, phosphoric acid, etc., and then combining with the gastric measures, when the stomach has recovered much of its tone, one of the least astringent preparations of iron—selection of the individual form being made in each case after careful consideration of all the surrounding circumstances, and guided by the results of actual trial.

Dilated Stomach.—As the symptoms of indigestion accompanying dilatation of the stomach are mainly those of delayed emptying of that viscus, with consequent stasis of and subsequent onset of fermentative processes in its contents, treatment must be directed towards removal or relief of such aberration of function, but must also take cognisance of the primary cause which has led to the dilatation, whether it is the sequel to pyloric obstruction, organic or spasmodic; be the result of myasthenia gastrica, with loss of resiliency of the muscular coat; of habitual over-filling of the organ throughout a prolonged period of time; of frequent over-distension in flatulent dyspepsia; or, again, the after-effect of repeated and protracted attacks of chronic catarrh. Little can be done to relieve the symptoms which are due to organic pyloric stenosis, save by regular removal of the stomach contents with the stomach tube, after every chance has been given for the absorption of all available nutriment through the stomach mucous membrane on each occasion; or by operative interference and excision of the constricted pylorus with formation of a gastro-duodenal intercommunication. Spasmodic closure of the pyloric sphincter is commonly the result of irritation from hyperacid contents of neurotic origin; and the gastric dilatation which sometimes follows repeated attacks of this form of contracture, may readily disappear on removal of the exciting cause, provided that hypertrophy of the pyloric tissues has not been induced to any great extent.

The treatment of *gouty indigestion*, particularly where the indigestion belongs to the chronic nervous variety, is fraught with many difficulties. The gastric and intestinal symptoms can rarely be treated save to the detriment of the gout; the gout, but with harm to the dyspepsia. In some it may appear advisable to deal directly with the digestive symptoms first; in others to influence them indirectly through the alleviation of the gouty habit. If the type of indigestion present permit, fractional dieting, mostly by milk, with copious flushings out of the organism with large amounts of plain water, or non-irritating mineral waters, such as Contrexéville, may benefit both factors.

Infant Feeding.

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See also Diet, Invalid Feeding

The importance of an accurate knowledge of the principles and practice of infant feeding can hardly be overestimated. The appalling mortality of infancy is due in no small degree either directly or indirectly to faulty feeding. Nor is this faultiness of feeding limited to the poorer classes, it is by no means uncommon amongst the wealthy, and it would seem to be due more often to ignorance than to lack of means

Of recent years much has been written concerning the physiology and chemistry of infant feeding, and there is a growing tendency in practice also to transfer the feeding of infants from the old rule of thumb to the region of exact science. In the present article the physiological and chemical aspects of the subject will be considered chiefly in their direct bearing upon the practical problems of infant-feeding, but it may be well to make some preliminary remarks with reference to these points.

In a general way it may be said that infants require the same food-constituents as adults. To repair the waste of tissue metabolism at any period of life the food must contain five elements, namely, proteids, hydrocarbons, carbohydrates, salts, and water. But the most elementary knowledge of the physiology of infancy shows that these constituents must be supplied in very different forms and proportions from those which can be used for adults or even for children over the age of two years.

During the first six months of life the teeth have usually not yet appeared, no food therefore which requires mastication, such as bread, potatoes, or puddings, can be suitable at that age, indeed in many cases, even at the age of twelve months, the teeth are not sufficiently advanced to deal with such articles of diet.

The saliva and pancreatic secretion only gradually acquire their full amylolytic power. The writer found that distinct though feeble amylolytic action was exerted by an infusion of the parotid gland from an infant still-born at full term; it would seem, however, that the saliva does not acquire any considerable degree of this power until the infant is three months old, while the pancreatic secretion which plays the most important part in the digestion of starch does not attain to its full amylolytic power until several months later, perhaps not until the end of the tenth or eleventh month. For this reason the carbohydrate element in food must not be supplied in the form of starch during the earlier months of life, it must be given in the form of sugar. The difficulty in digesting such articles as bread, potatoes, or any of the numerous so-called "infant-foods" which contain starch, can therefore readily be understood. The gastric juice which plays the chief part in the digestion of proteids has its peptonising powers well developed from the time of birth, so physiologists tell us; but experience shows that these powers are but feeble compared with those in older children, and that any but the most easily digestible of proteids quickly overtax the infant's stomach. This is particularly noticeable in the difficulty which infants find in digesting the casein in cow's milk. But here another peculiarity of early life has to be considered. The thinness and weakness of the muscular wall of the stomach and its feeble contractions render peristalsis less effective than in later life, so that solid masses, such as are formed by the casein of cow's milk, may be less thoroughly mixed with the gastric secretion, and therefore less easily digested.

But there are other considerations which determine to some extent the diet of infancy. Metabolism at this period is altogether more rapid than in later years; infancy is *par excellence* the age of construction. Never again does anything occur comparable to the extraordinary rapidity of growth in infancy. That an infant should actually double its weight in five months and treble it in twelve is evidence of the marvellous activity of tissue growth at this period. To supply material sufficiently rapidly, not merely to compensate for the waste which accompanies metabolism, but also to serve for the building of the growing structure, it is necessary to feed much more frequently than in later life, and to give the food in such a form that it shall be capable of ready absorption and assimilation. The most perfect fulfilment of these requirements is seen in the mother's milk, with its finely divided curd, so easily acted upon by the gastric juice, its comparatively large proportion of soluble proteid, and still larger proportion of soluble carbohydrate.

The importance of a large proportion of fat in the diet corresponds to the vital necessity for warmth at this age. To supply this warmth the infant requires a considerably larger proportion of hydrocarbon, the heat-producing element in the food, than adults do. Physiological observations have shown that while for an adult the proportion of fat to proteid required is about 1 to 3, for infants it is nearly 2 to 1. But the need of warmth is certainly not the only reason why more fat is required for infants than for adults. It would seem that fat, taken as such in the food, plays an important part, directly or indirectly, in the growth of the tissues; in particular there is strong evidence to show that if the food be deficient in fat the growing bone is very apt to show rickety deformities.

HUMAN MILK

The only food which fulfils all the requirements of early infancy is human milk, and the nearer any other food approximates in its character to human milk the more suitable it is for the feeding of infants.

Human milk is an opaque, yellowish-white fluid, with an average specific gravity of 1030-1032 (the writer found variations from 1027 to 1034). It is neutral or faintly alkaline in reaction. Its taste is sweet. It is usually sterile, but recent investigation has shown that this is not always so, the staphylococcus aureus and albus are sometimes found in it, and it seems probable that these and other bacteria may find their way into the ducts from the outside.

Microscopic examination shows that the milk consists of two parts: (i.) A colourless plasma, which contains proteids and salts in solution; (ii.) Globules of fat suspended in the plasma. The proteids found dissolved in the plasma are chiefly of two kinds: (1) Casein, or more accurately speaking, caseinogen, which is precipitated by acids, and also by a special ferment, rennet, in the gastric juice; (2) Lactalbumen, which is not precipitated either by acids or by rennet, but is coagulated by boiling. The fat globules in human milk measure from $\frac{1}{3000}$ to $\frac{1}{12,000}$ inch in diameter.

Many analyses of human milk have been made with widely-varying results. The practical difficulty is great, for unless the whole contents of the breast are used no strictly accurate result can be obtained, and it is not easy to secure the whole of the milk. The discrepancies between various observations are no doubt due partly to this fact, for the composition of the milk varies according to the part taken for examination. The milk may be divided into three portions, the fore-milk, the middle-milk, and the after-milk or strippings. An examination of the fat alone will show how widely these may differ in composition. In a specimen of fore-milk the writer found 1·4 per cent of fat, in one of after-milk 10 per cent of fat; in one case the fore-milk showed only ·4 per cent of fat, while the middle-milk showed 3·2 per cent. The proportion of proteid, similarly, is found to increase gradually, being lowest in the fore-milk and highest in the after-milk. It is obvious, therefore, that to obtain the average proportions of the various constituents a mixed sample of the whole contents of the breast should be taken. In practice this is seldom possible, and the next best course is to use only the middle-milk, from which presumably a fair average estimate can be obtained. A convenient method is to inquire how long the infant usually takes to empty the breast, and then to direct that it shall be put to the breast for one-third of this time, usually about five minutes, after which the specimen of milk, which should not be less than one ounce, is drawn off for analysis.

The average composition of human milk, calculated from various published analyses, may be stated as follows:—

			Variations.
Proteids	Casein ·6 per cent Lactalbumen 1·4 per cent	2·0 per cent	(1-4·8)
Fat		3·5 „ „	(2·4-4·2)
Lactose		7·0 „ „	(5·8-7·4)
Salts		·2 „ „	(·1-·37)
Water		87·3 „ „	(86·7-88·5)

Colostrum.—During the first two or three days after parturition, before the flow of milk is established, the mother's breast yields a fluid, colostrum, which has a somewhat different composition. It contains less fat and sugar, but a larger proportion of proteid. Microscopically, also, it shows certain large, round, or ovoid granular bodies, the "colostrum corpuscles," which are three or four times as large as the fat globules in later milk. In addition to its food-value the colostrum has been thought to have some laxative effect; this is certainly not due to excess of fat; in ten samples examined by the writer the average percentage of fat was 2·4, and some contained only 1·0 or 1·2 per cent. The curd formed with an acid was in some of these cases much firmer and larger than is usually found in ordinary human milk.

Breast-Feeding

Importance of Breast-feeding.—No artificial infant-feeding can ever be so satisfactory as nature's method of suckling at the mother's breast. One can hardly condemn too strongly the selfishness of those women who refuse to feed their infants in the natural way because suckling interferes with their social pleasures or engagements. From some statistics collected by the writer at the Hospital for Sick Children, it would seem that 90 per cent of the fatal cases of diarrhœa in infancy occur in those who are being partially or entirely hand-fed, only 10 per cent are in infants fed entirely at the breast.

Artificial feeding is responsible for the vast majority of the cases of gastro-intestinal disorder in infancy, while infantile scurvy and rickets are pre-eminently diseases produced by artificial diet, and that too in many cases where considerable care has been exercised in the feeding of the infant. Even where a scanty secretion of milk or other circumstances render it impossible for the mother to feed the child entirely at the breast, it is most important that she should continue partial breast-feeding, for it has been shown that even partial suckling considerably diminishes the risk of fatal diarrhœa, and also the liability to other disorders which are due to artificial feeding.

Conditions which contra-indicate Breast-feeding.—In many cases arrest of the milk secretion necessitates feeding by hand, but there are other conditions which may make it advisable to wean the infant.

General debility in the mother is sometimes a sufficient reason for weaning the child; too often, however, this is merely an excuse for getting out of a disagreeable duty, and because a mother says she "never could suckle her children," it by no means follows that she cannot suckle her present infant; the medical man must judge for himself whether the general ill health is sufficient to justify partial or complete weaning of the infant.

It occasionally happens that the mother's milk persistently disagrees with the infant. The reasons for this will be considered below; here it need only be pointed out, that in many cases by proper regulation of the mother's habits and diet it is possible to correct the fault in the milk, and that until such an effort has been made the child should not be weaned.

Tuberculosis in the mother makes it unadvisable for her to suckle her infant. If one may judge from the experimental evidence as to tuberculous cows the risk of infection is extremely small, unless the breast itself is tuberculous, but so long as there is any active tuberculosis in the body the possibility of infection by the milk cannot be absolutely excluded, and, moreover, the activity of the disease in the mother seems in some cases to be increased by suckling, so that it is wiser on the whole to wean. The presence of old tuberculous lesions, such as spinal curvature or scars of tuberculous glands, so long as they are quiescent and there is no active focus of infection elsewhere, need not prevent the mother from suckling her infant.

Syphilis in the mother does not *per se* contra-indicate suckling, except in the very rare cases where syphilis is acquired during pregnancy and the child shows no evidence of syphilitic taint. Where the mother shows no symptoms of syphilis, but the infant shows evidence of the inherited disease, there is some doubt as to whether suckling should be allowed. Apparent exceptions to Colles's law have occurred, in which the infant has infected the mother in this way, but such cases are extremely rare, and the vital importance of breast-feeding, especially for the weakly syphilitic infant, has to be balanced against the very remote chance of infecting the mother.

The risk of infection is probably much greater for a wet nurse, who certainly should never be allowed to nurse such an infant.

Infants have occasionally acquired syphilis after birth; the risk of infection from such infants is great, and necessitates immediate weaning.

The occurrence of an acute febrile disease on the mother, if it last more than two or three days, may necessitate taking the child from the breast, partly because the milk under these conditions sometimes disagrees with the child, and partly for the mother's sake; but it is well to remember that even after the child has been kept from the breast for two, three, or even four weeks, it is sometimes possible to start the flow of milk again by putting the child back to the breast, and in some cases the secretion will continue if the milk is drawn off by a breast-pump regularly during the mother's illness. Often, however, the milk ceases altogether if suckling is stopped, even for a few days; it is important, therefore, not to take the child from the breast unless it becomes absolutely necessary. Puerperal fever or any other of the infectious fevers necessitate weaning.

The return of the menses during lactation is no reason in itself for weaning. An idea seems to be prevalent that when this occurs the milk necessarily becomes harmful to the infant. Undoubtedly the milk does sometimes disagree with the infant during the menstrual period but even where this occurs the disturbance is usually only transitory and slight, and more often there is no disturbance whatever. That some variation may occur in the composition of the milk during menstruation has been shown by analysis, in some cases the proteids and fat have been increased, in others diminished; but in most cases the variation is so slight as to produce no evil effect.

Pregnancy occurring during lactation does not necessarily call for immediate weaning. It seldom happens that pregnancy occurs before the infant is some months old, and lactation can usually be prolonged with safety for three or four months after the mother becomes pregnant; it is generally wiser not to continue the suckling longer than this, especially where the mother has had previous miscarriages or is in feeble health. If the quality of the milk seems to be poor the infant should be weaned. Dr.

Cheadle states that the only case in which he has seen rickets occur in an infant at the breast was one in which pregnancy occurred during lactation.

Failure of nutrition or symptoms of gastro-intestinal disturbance in the infant are considered last in this connection, for it is rarely indeed that they contra-indicate breast-feeding. One of the commonest of complaints is that the mother's milk "does not seem to satisfy the child," and mothers sometimes suggest weaning, or more often the addition of artificial food, on this account. Such a complaint should never be considered adequate reason for premature weaning. In many such cases the apparent hunger of the infant is really the result of dyspepsia from too frequent, too greedy, or too irregular feeding, and the child becomes more content when the breast-feeding is properly regulated; in other cases the milk is scanty, but by judicious dieting of the mother it may be possible to increase the supply; in others again, but these more rare, the composition of the mother's milk is at fault, and if it cannot be altered by proper regulation of the mother's habits it may be necessary to wean the infant. These points will be considered more fully below, here it will be sufficient to say that even partial artificial feeding is not to be recommended unless absolutely necessary, and in all such cases the surest guide is the progress of the infant's weight; complete weaning is only very rarely to be recommended in such cases.

Directions for Breast-feeding.—An infant should be put to the breast within three or four hours of birth; by the mere action of sucking it stimulates the mother's uterus to contract, and the possible laxative action of the colostrum apart from its nutritive value may be beneficial. As a rule, for the first two or three days the very small amount of colostrum which is obtainable is quite sufficient, but when the infant is very feeble it is sometimes advisable to give it other food; for this purpose whey may be given from a spoon (a dessertspoonful or tablespoonful every three or four hours) until the secretion of milk is established. Infants will sometimes refuse the breast altogether owing to the mother's awkwardness in giving it; it may therefore be necessary to explain the manner of nursing, especially to a young mother. The child should be held on the arm of the same side as the breast which is to be used, in a comfortable semi-recumbent position, with the head slightly raised and the body tilted slightly towards the mother, who should bend slightly forward. The nipple should be supported between the index and the middle fingers of the disengaged hand, which can thus be used either to control the flow of milk by squeezing the nipple between the fingers, or to assist the flow by compressing the breast with the palm of the hand.

Depressed nipples may give considerable trouble. The putting of the infant to both breasts shortly after birth while they are still flaccid before the milk secretion begins, allows it to grasp the nipple and pull it out more easily than it could when the breast is tense; but in many cases artificial suction is necessary, and if this fail, and the depression is such as to prevent the child from sucking, it is worth while to try whether sucking can be accomplished through a rubber nipple attached to a glass breast-shield. As a last resource the milk may be drawn off by a breast pump, and given to the child in a spoon or bottle as long as the milk can be obtained thus.

The breasts should be used alternately; one breast contains, as a rule, sufficient milk for one meal. The infant should take about fifteen minutes to empty the breast; it should never be allowed to fall asleep with the nipple in its mouth. It should be accustomed from the beginning to lie quietly in its cot after feeding: many infants will vomit if jolted in the mother's arms just after a meal.

The nipple after feeding should be washed with warm water and carefully dried.

Frequency and Regularity of Breast-feeding.—During the first two days after birth, before the milk secretion is established, the infant should be put to the breast every four hours. After this the infant should be fed as follows:—

Age.	Interval between Meals.
First week to end of first month	2 hours.
End of first month to end of third month . . .	2½ "
End of third month until weaning	3 "

Even from the beginning it is important to accustom the child to miss one feed at night, so that a period of at least four hours' uninterrupted rest may be ensured to the mother. This interval without food can gradually be increased, until at the age of three months the infant will sleep as long as six hours at night, the last feed being given at 11 P.M., and the first morning feed at 5 A.M. After the age of six months a healthy infant may be allowed to sleep for as long as seven or even eight hours at night if it will, without being disturbed for food.

One is sometimes asked the question, whether an infant should be disturbed during the day if asleep at the time when its feed is due. Undoubtedly it should: regularity in the time of feeding is most important both for the mother's sake and for the child's, and the disturbance of sleep is of little consequence, as an infant usually falls asleep again very quickly after it has been fed. It is quite easy to accustom an infant to expect its meals at regular intervals as timed by the clock; indeed, some infants will wake almost to the minute when their feed is due, if only these regular habits are insisted upon from the very beginning. Unfortunately the habit is easily broken, and on this account the rule of regularity in infant feeding must be as the laws of the Medes and Persians. Some of the commonest troubles of infancy—such as flatulence, colic, hiccough, fretfulness and sleeplessness—are due to neglect of this rule.

Some writers have advocated the use of one artificial feed daily on the ground that circumstances may occasionally make it impossible for the mother to suckle at the proper time, and that if the infant is accustomed to take food from the spoon or bottle a difficulty is thus avoided, and also that it facilitates weaning. In the present writer's opinion such artificial feeding as a routine practice is certainly not to be recommended, it encourages the mother to think lightly of the importance of breast feeding, in fact it facilitates neglect of her duty; it introduces a very serious element of risk which is totally unnecessary; and, lastly, any real difficulty in weaning is so exceptional that it may be disregarded.

VARIATIONS IN HUMAN MILK

Quantity.—According to some observations quoted by Dr. Holt, it would seem that the average quantity of milk secreted in the twenty-four hours increases gradually during lactation from 10-16 oz. at the end of the first week, up to 30-40 oz. at the sixth to ninth month. The amount taken by the infant at each feed also increases from ⅝ to 1½ oz. during the first week up to about 3 oz. at the end of the first month, and then varies from 2 oz. to 5 oz. at the end of the third month, and from 3 oz. to 7 oz. from the third month onwards. The accurate measurement of the milk secreted by the mother, and also of that taken by the infant, is no easy matter. The latter may be determined roughly by weighing the infant immediately before and after each meal; the total amount calculated thus for the twenty-four hours gives approximately the quantity secreted in that time by the mother. A more direct and accurate method of determining the daily secretion would be to draw off the contents of the breasts at intervals during the day and measure them, but this is hardly ever feasible. Fortunately for practical purposes, it is very rarely necessary to make any actual measurement of the milk, for any marked deficiency can hardly escape the mother's observation, and is sometimes obvious to any one who watches the infant at the breast—it sucks vigorously for a few minutes, and then failing to obtain milk, rejects the nipple and cries out peevishly; in other cases it remains sucking much longer than it should, perhaps as long as half an hour or more, evidently getting the milk very slowly.

Deficiency of Milk.—Even if there is reason to think that the quantity of milk secreted is below the average, it must not be hastily concluded that the addition of artificial food is necessary. The quantity may be quite sufficient for the needs of the infant, and the most reliable criterion of this sufficiency, as already pointed out, is the weight of the infant; so long as this is increasing it is not only unnecessary but unwise to make any addition. Where the breast milk is insufficient it may be increased by the mother's taking milk and gruel freely. In some cases the same result may be obtained by taking malt extract.

Malted liquors certainly increase the flow, and some mothers are enabled to feed their infants by taking half a pint of good beer or stout daily; this is, however, quite unnecessary as a rule, and should never be ordered without special reason, for apart from other obvious objections, the drinking of such a beverage by the mother sometimes alters the character of the milk so that it disagrees with the infant.

Excess of milk is quite uncommon, and but rarely calls for treatment. The fluids in the mother's diet should be restricted, and if there is any sign of the breast becoming unduly hard or tender, some of the milk should be drawn off two or three times a day in the intervals between the infant's feeds.

Quality.—The proportion of each constituent of human milk is subject to some variation, and this is noticeable even in samples taken from the same mother at short intervals. The sugar and salts would seem to be more constant than the others, and their slight variations are of no practical importance. The fat and proteids show considerable variations which are of great importance, for excess or deficiency of either of these constituents may render the milk unsuitable for the infant.

The actual proportions of each constituent can only be determined by analysis. Unfortunately there is no simple method available for clinical purposes in the estimation of the proteids, but a rough estimate can often be made from the specific gravity if the proportion of fat is known. The sugar and salts may be considered as practically constant, and therefore any variation in specific gravity depends upon the fats or the proteids. The higher the proportion of fat the lower is the specific gravity, and consequently, if with a high proportion of fat the specific gravity is high the proportion of proteids must be above the average; if with a low proportion of fat the specific gravity is low, the proportion of proteids must be below the average. If the proportion of fat is normal, then the specific gravity will vary directly as the proportion of proteids.

For determining the specific gravity of breast milk it is convenient to use glass beads specially made to float in fluids of certain specific gravity; in this way, with an ordinary test-tube or small conical medicine glass it is possible to determine the specific gravity where only a few drachms of breast milk can be obtained. It may be repeated here that only the middle-milk must be used for any such estimation of the quality of the milk, for as mentioned above, an entirely erroneous estimate will be obtained if either the fore-milk or the after-milk is used.

The fat may be estimated by the following simple method:—A cylindrical five-cubic centimetre measure, having a diameter of $\frac{3}{8}$ inch, and graduated in cubic centimetres and millimetres, is filled with the breast milk exactly up to the five-cubic centimetre mark; it is then corked and allowed to stand in a warm room (65° F) for twenty-four hours. The cream rises to the top and is read off in cubic millimetres. Empirically the cream readings are found to correspond with fat percentages approximately thus:—

Cream.		Fat.
2 cub. millim.	=	2·8 per cent.
3 " "	=	3·5 " "
4 " "	=	4·4 " "
5 " "	=	5·6 " "

The method gives only a rough approximation: the ease with which cream rises depends on several varying factors, so that two samples of breast milk containing an equal quantity of fat may show a slight difference in the depth of the cream. As a simple clinical method, however, this may be useful for determining whether the milk is poor or rich in fat. Any milk which contains less than 2 cub. millim. of cream may certainly be considered deficient in fat.

The commonest and most serious defects in breast milk are excess of proteid and deficiency of fat. Upon one or other of these two faults depend many, perhaps most of the gastro-intestinal disorders of infants at the breast. It is a matter of the greatest practical importance in any case where an infant is not thriving at the breast to determine which constituent if any is at fault; too often weaning is recommended without any investigation of the milk, or without any attempt to rectify a fault which can sometimes be corrected by very simple means.

Excess of proteid is of course a relative term: the amount of proteid which can be digested varies with different infants. Excess may be suspected where the stool is pale, as if from undigested curd, and therewith the infant suffers from colicky pain, constipation, and perhaps vomiting of curd. Such a condition of milk may be habitual to a particular woman, and may make it almost impossible for her to nurse her infant; more often, however, it is an induced condition, and probably depends in many cases on too frequent nursing. It has been found that the proteids in cow's milk are increased by increasing the frequency of milking, and from clinical experience it would seem that the same result occurs in human milk. So that excess of proteid may sometimes be reduced by lengthening the intervals between the feeds. In other cases an inactive life together with a diet too rich in proteid food is responsible for this condition of milk. Menstruation is sometimes the cause of a temporary increase in the percentage of proteids.

Any defect in the mother's habits or diet must be remedied, and if after this the infant is still unable to digest the proteid in the milk, the difficulty can sometimes be overcome by giving a tablespoonful of plain water or of barley water to the infant immediately before nursing; this seems to dilute the proteid, and in some cases entirely obviates the difficulty. If this fails, the administration of a small dose of sod. bicarb (gr. ij.) with papain (gr. j.) immediately before feeding sometimes proves successful.

Deficiency of proteid seldom occurs apart from deficiency of fat, and when both are deficient the clinical results are generally due rather to the lack of fat than to the lack of proteid. Such a poor milk generally has a watery appearance, and is very apt to produce troublesome flatulence; the child may cease to gain weight, and may even waste although the flow of milk is abundant. In such cases the mother has sometimes been poorly fed, and the milk may be improved by increasing the proteids in the diet, and by attention to the mother's general health.

Excess of fat is quite uncommon and hardly ever produces any bad symptoms; but the writer has thought that occasionally troublesome vomiting may have been produced thus. A reduction of the quantity of food, especially of meat and eggs, taken by the mother may reduce the percentage of fat.

Deficiency of fat is much less rare, and may occur alone or in association with deficiency of proteid. Infants fed on such milk fail to increase in weight as they should, they suffer from chronic constipation and its attendant troubles, and may even show signs of rickets. It is often possible to detect any considerable deficiency of fat by a mere inspection of the milk, which has a watery, grayish appearance, but this of course gives only a very rough idea of the degree of deficiency. In any case where there is reason to suspect that the infant is suffering from such a deficiency of fat, an accurate analysis of the milk should be made if possible, or the fat may be estimated by the simple method suggested above. Human milk should certainly show not less than 3 per cent of fat, and any proportion lower than 2 per cent is likely to be associated with some disorder or failure of nutrition in the infant. If it is found by repeated estimations that the proportion of fat is continuously low, an attempt should first be made to increase it by increasing the proteids in the mother's diet. For this purpose meat, poultry, or fish, and oatmeal porridge are valuable, and at the same time eggs and milk should be taken freely. As far as possible worry is to be avoided, and work or recreation must always stop short of fatigue. If these measures fail to increase the proportion of fat the deficiency must be made up by giving the infant two or three feeds daily of hot water and cream, or whey and cream (see Artificial Feeding), or if this is not convenient, cod-liver oil in doses of 10 to 20 drops according to the age of the infant may be given two or three times a day.

Deficiency of fat is not to be regarded as a reason for weaning: fat can readily be supplied in an easily digestible form by supplementing the mother's milk as suggested, whereas if the child is weaned entirely it becomes necessary to supply proteids also, and experience shows that one of the chief difficulties in artificial feeding is to supply the proteids in a digestible form.

Conditions in the Mother affecting the Milk.—The effect of menstruation and of pregnancy on lactation has already been considered. The milk may be diminished in quantity or lost altogether as the result of worry or mental shock. The quality of the milk is also sometimes seriously affected by any strong emotion, such as fright, anger, or grief, and under these conditions the milk may cause severe gastro-intestinal disturbance in the infant. Where this occurs the infant should be fed artificially for one or two days if necessary, and then again tried at the breast. In some very emotional women the milk fluctuates so frequently in quality that it may be necessary to wean altogether.

Effect of Drugs.—Certain drugs are found to be excreted partly in the milk, and may thus affect the infant. Of these the most important are belladonna and atropine, potassium iodide and bromide, salicylates, arsenic, and saline purgatives. There is some evidence that opium and morphia given to the mother may affect the infant, and in one case a fatal result has been recorded. Other drugs have occasionally been found in the milk, but not in sufficient amount to be of any practical importance.

The administration of drugs to the mother for the sake of their action on the infant has little if any advantage, and has very obvious disadvantages; besides the unpleasantness to the mother, the effect on the infant is very uncertain, and there is no means of accurately regulating the dose which the infant obtains; it is far simpler and more effectual to give the drugs to the infant in the usual way.

Weaning

Time of Weaning.—This must be determined chiefly from the circumstances of the individual case. As a rule an infant should not be weaned earlier than the end of the ninth month, nor later than the end of the twelfth month. It is very unadvisable to wean during the hot weather; in the writer's opinion it is often better to continue suckling even as late as the thirteenth or fourteenth month, if in this way weaning during the hot weather can be avoided. It is wise, also, not to wean at a time when teething is causing much disturbance; weaning should be done if possible in an interval between the cutting of teeth. Any transitory disorder, gastro-intestinal or otherwise, is usually sufficient reason for postponing the time of weaning. The conditions which may call for premature weaning have already been considered.

Method of Weaning.—Weaning may be sudden or gradual. The sudden cessation of suckling is hardly ever to be recommended, except in those cases where the obstinate refusal of the infant to take artificial food as long as it is suckled makes it necessary to stop breast-feeding altogether. Gradual weaning is more satisfactory in every way. The breast feeds are gradually replaced by artificial feeds, until at the end of four or five weeks all the meals consist of artificial food. The infant in this way gradually becomes accustomed to its altered diet, and digestion is much less likely to be upset. Many infants wean themselves: sometimes quite suddenly they seem to take a dislike to the breast, and refuse to suck; in some cases it is only one breast which the child refuses, and partial or entire suckling may be continued with the other. This refusal often occurs at a time when the milk is becoming scanty, at other times no cause whatever for the sudden dislike can be found.

Wet-Nursing

Where the mother is unable for any reason to suckle her infant the question arises whether a wet-nurse should be employed. There can be no doubt that, so far as the nature of the food goes, the only perfect way of replacing the mother's milk is by a wet-nurse; but the objections to this method are so very serious that it is now comparatively seldom used. The neglect of the nurse's own infant, possibly at the cost of its life; the immoral character of some of the women thus employed; the difficulty of excluding the presence of syphilis in women of whose previous history nothing is known;—these are some of the weightier objections, to which have to be added such minor points as the inconvenience to the household which the presence of such a nurse often involves, and the costliness of such a method of feeding.

If it is decided to employ a wet-nurse she must first be thoroughly examined by the medical man, particularly for the presence of tubercle or syphilis. Quite recently the writer had under his care an infant whose wet-nurse had deliberately concealed the fact that she had hæmoptysis, and although she had been examined by a medical man, it was not until she had suckled the infant several months that the fraud was discovered by her becoming acutely ill with tuberculosis. The nurse's infant should also be examined if possible, partly to see whether it shows any evidence of syphilis, and partly to ascertain whether it has thriven on its mother's milk.

In the choice of a wet-nurse, her age and the age of her infant are of much less importance than the character of her milk, and this should be ascertained by the clinical methods described above, or better still by having an accurate analysis made. Inspection of the milk and also of the breasts, which should be full and firm, will also give some idea of the quantity and quality of the milk.

The diet of a wet-nurse must be regulated on the same lines as those laid down for the nursing mother, but with this caution, that as she has usually come from very poor circumstances, a too plentiful or too rich diet may easily alter the quality of her milk, so that it may disagree with her nursling. For the same reason, if she has been accustomed previously to hard, active life it is important that she should have plenty of exercise, otherwise the combination of a liberal diet with insufficient exercise is almost sure to produce milk too rich in proteid and fat.

The employment of a wet-nurse is by no means always a success; not every woman's milk agrees with every infant, and even if at first the nurse's milk agrees it is liable to any of the changes to which a mother's milk is subject, and may at any time cease to agree with the infant; and lastly, such women require constant supervision to see that they discharge their important duties faithfully.

Artificial Feeding

When the mother is unable to suckle her infant, the most readily available, and in most cases the best substitute for breast-feeding is cow's milk; but as will be seen, cow's milk requires modification to adapt it to the needs of the infant, and to carry out this modification intelligently some knowledge of the composition of cow's milk, and of the points wherein it differs from human milk, is essential.

Cow's milk, when first drawn from the udder, has an amphoteric reaction, but as it reaches the consumer it is generally faintly acid. Its specific gravity varies from 1029 to 1035. Microscopically it resembles human milk in consisting of a colourless plasma in which are suspended fat globules: the average size of these globules, however, is larger than in human milk. The plasma, as in human milk, contains proteids, sugar, and salts in solution, but the proportions are not the same as in human milk.

The composition of cow's milk varies considerably: the following average obtained from analyses made by several observers is therefore only approximate; the proportions in human milk are given here for comparison :—

Cow's Milk.		Human Milk.	
Proteids : Casein, 3·25 Lactalbumin, ·75	4·0 per cent	2·0 per cent	Casein, ·6 per cent Lactalbumin, 1·4 per cent
Fat	3·5 „ „	3·5 „ „	
Sugar	4·0 „ „	7·0 „ „	
Salts	·7 „ „	·2 „ „	
Water	87·8 „ „	87·3 „ „	

It will be seen that the chief differences in composition are the much larger proportion of proteids and the much smaller proportion of sugar in cow's milk. But the proteids show an even more important difference, for while in human milk they consist chiefly of lactalbumin, in cow's milk they consist chiefly of casein: the proportion of casein to lactalbumin in cow's milk is about 4 : 1, in human milk it is about 1 : 2. The practical significance of this difference is readily appreciated if samples of the two kinds of milk are taken in two test-tubes, and some weak hydrochloric acid is added. Large solid masses of curd are at once formed in the cow's milk, while the human milk shows only very fine flocculent shreds of curd floating in it. Similar changes occur in the stomach, but the formation of curd here is due not only to the acid in the gastric juice, but also to a milk-curdling ferment, rennet. Another important difference is the sterility of human milk in contrast with the large number of bacteria present in cow's milk as it reaches the consumer; the alkalinity of human milk also contrasts with the acidity of cow's milk.

Modification of Cow's Milk.—It is obvious that in order to reduce the proportion of proteids to the standard of human milk some dilution will be necessary. For this purpose various diluents are available, but the choice of the diluent in any particular case is often a matter of considerable importance.

Plain water, as the simplest and least expensive, is most commonly used; such simple dilution not only reduces the percentage of proteid, but also materially reduces the size of the curd. In many cases this is all that is required to enable the infant to digest the casein; in others, however, this is not sufficient, and the digestion of the curd may be assisted by using some thickened fluid as a diluent.

Barley water is often used in this way, and whatever may be the rationale of its use, there can be no doubt that some infants will digest milk diluted thus when they are unable to digest milk diluted with plain water. It may be that such a thickened fluid does to some extent mechanically hinder the formation of large curds, but from a series of experiments made by the writer, it would seem that this attenuating action, if present at all, is extremely slight, and it has been denied altogether by some observers.

As a food in itself barley water has but little value; its most important constituent is starch, and although this is present in very small proportion, it sometimes causes trouble, especially in the earlier months of infancy. Flatulence and colic, and occasionally diarrhœa, have seemed in the writer's experience to be due to the use of barley water as a diluent. On the other hand, the laxative effect of barley water may be useful for an infant who is suffering from habitual constipation. Rice water and oatmeal water are sometimes used in the same way. The former has less laxative action than barley water, and may therefore be useful where there is a tendency to diarrhœa. Oatmeal water, although it has a distinct laxative effect, contains less starch than barley water, and may agree where barley water has failed. Gelatine is occasionally used in the form of a thin jelly, in the proportion of one teaspoonful to about four ounces of milk and water.

The methods of preparation of these diluents are as follows:—

Barley water is made by putting two teaspoonfuls of pearl barley into a pint of cold water, boiling slowly down to two-thirds of a pint, and then straining; or by mixing one teaspoonful of prepared barley (in powder) with one pint of boiling water, and then boiling for ten minutes. Whichever way it is made, a fresh supply must be prepared twice a day, and it must be kept in a cool place.

Rice water is made by soaking one tablespoonful of rice, which has previously been washed with cold water, in a quart of water at tepid heat for three hours; it is then boiled slowly for an hour, after which it is strained.

Oatmeal water is made by mixing one tablespoonful of oatmeal with a pint of boiling water, and boiling slowly for one hour; water must be added as necessary to make up the quantity after boiling to one pint; then strain.

Gelatine jelly is made by soaking one teaspoonful of gelatine in a teacupful of cold water for three hours, then standing it in hot water which is kept boiling until the gelatine is dissolved.

Another method which is occasionally useful, and which, perhaps, has some mechanical action as suggested above, is to add to the milk, after dilution with plain water, a very small quantity of some malted preparation of cereals, such as Mellin's food; half a teaspoonful or one teaspoonful of this added to the feed of milk and water will sometimes enable an infant to digest the casein of the milk.

Lime-water may often be used with advantage instead of, or in addition to plain water. This not only serves to dilute the proteids, but also seemed, in some experiments made by the writer, to have a definite though slight effect in diminishing the size and firmness of the curd; it also corrects the acidity of the cow's milk, and undoubtedly increases its digestibility. Lime-water should be used in the proportion of at least one tablespoonful to a three-ounce mixture of milk and water. A solution of sodium bicarbonate (gr. iv. to ℥j.) may be used similarly; it has the advantage that it does not cause constipation as lime-water often does. For this very reason, however, lime-water is usually to be preferred as a diluent in cases of diarrhœa, and in such cases two tablespoonfuls may be given in a three-ounce feed. The more concentrated form of lime-water known as liquor calcis saccharatus is sometimes more convenient; a teaspoonful of this to four tablespoonfuls of water gives a solution of nearly the same strength as the ordinary lime-water. A simpler way of using it is to add the saccharated lime solution directly to the food, for instance, to a three-ounce feed of milk and water fifteen drops of the liquor calcis saccharatus should be added.

The addition of an equal quantity of any of the diluents mentioned will reduce the proportion of proteids in cow's milk to 2 per cent, as in human milk; but the relative proportions of casein and lactalbumin remain exactly as before, so that the casein is still much more than in human milk; this can be seen from the comparison below. Experience shows that it is this excess of casein, and the large solid curd resulting therefrom, which is the chief difficulty in the use of cow's milk for infants' food, and in many cases an infant will not digest cow's milk until the dilution is carried so far that the *casein* is reduced to about the same percentage as in human milk,—in other words, the cow's milk has been diluted with three or even four times its quantity of the diluent.

Whatever dilution is used it is evident that all the other constituents of the milk are diluted at the same time, so that the percentage of fat which was previously the same as in human milk becomes too low, and the already insufficient proportion of sugar becomes even less sufficient. This may be seen from the following figures:—

Cow's Milk diluted with an equal Quantity of Water.		Human Milk.	
Proteids : Casein, 1·63 Lactalbumin, ·37	2·0 per cent	2·0 per cent	Casein, ·6 Lactalbumin, 1·4
Fat	1·75 „ „	3·5 „ „	
Sugar	2·0 „ „	7·0 „ „	

It is necessary, therefore, not merely to dilute the cow's milk in order to reduce the percentage of proteids, but also to add fat and sugar in order to bring the proportion of these up to the standard of human milk.

Addition of Fat.—The supply of a proper proportion of fat is a matter of the greatest importance in the feeding of infants. One of the commonest faults in infant feeding at the present time is the use of food containing too little fat, and many cases of chronic constipation, failure of nutrition, and rickets are due to this deficiency. Taking human milk as the standard, one would say that 3-4 per cent is the proper proportion, and although some infants will make satisfactory progress on less than this, it seems probable that 3 per cent is the minimum which should be allowed; certainly a proportion of fat below 2 per cent at any period during the first year of life is often associated with some disorder of nutrition.

If cream containing a known percentage of fat can be obtained it is a simple matter to correct the deficiency of fat in the milk. In towns where centrifugal machines are used in the separation of cream, it may be possible to obtain cream which contains a fairly constant average percentage of fat. Observations by the writer showed that fresh cream, as supplied by London dairies, contains on the average about 48 per cent of fat; but even such cream showed considerable variation (36·8-54 per cent) in its proportion of fat, and it is much to be desired that a standardised cream should be more generally obtainable. If the cream used contains 48 per cent fat, it is evident that one teaspoonful in a three-ounce feed will raise the fat 2 per cent; so that if used with a three-ounce mixture of equal parts of cow's milk and water, it will raise the proportion of fat from 1·75 to 3·75 per cent. Any desired percentage of fat can be obtained by similar calculation.

Cream is the upper portion of milk into which the fat globules have risen either (1) by gravity, that is, the milk has been allowed to stand until more or less of the fat has risen to the top, which has then been skimmed off as "cream," or (2) by centrifugalisation. The other constituents of milk remain practically unaltered, both in quality and quantity in the cream, and therefore, to be strictly accurate, it would be necessary to take into account the proteids and sugar of cream when we add it to the infant's food; for practical purposes, however, the quantity of the centrifugalised cream used is so small that in most cases the relatively minute proportion of those other constituents can be neglected, and only the fat in the cream needs to be considered.

Perhaps the simplest way to obtain a standardised cream is to prepare it at home by allowing a quart of milk to stand for three hours in a cool place, in a glass water-bottle such as can be obtained from any scientific instrument maker for three or four shillings, with a tap near the bottom of the vessel. After the milk has stood for three hours the lower portion is drawn off, and can be used as skimmed milk for domestic purposes, the upper part into which the fat has risen, the cream, is used for the infant. The percentages of fat thus obtained can be seen from the following table; they are only averages, for, as already mentioned, the rate at which fat rises depends on several varying factors; but these figures are sufficiently accurate for practical purposes, they were obtained with average London milk:—

Quantity of Upper Milk (Cream) from each quart of milk.	Proportion of Fat.
One pint	5·5 per cent
Fifteen ounces	6·5 „ „
Ten ounces	8 „ „
Five ounces	11 „ „

Such top-milk or cream can be used either as an addition to a mixture of milk and water to increase the fat, or more simply with direct dilution with water. Thus the addition of a tablespoonful of the 11 per cent cream to a three-ounce mixture of equal parts of milk and water, will raise the fat percentage from 1·75 to about 3·2; or the dilution of one part of this 11 per cent cream with two parts of water will give a fat percentage of about 3·7.

One great drawback to the use of mixtures which contain added cream is the difficulty, especially in towns, of obtaining it fresh. Amongst the poor also the expense makes it impossible in many cases to obtain cream.

A plain mixture of milk and water is therefore commonly used, and the results are sometimes quite satisfactory; but in a considerable proportion of cases the inevitable deficiency of fat in the mixture leads to some failure of nutrition, or to some degree of rickets.

Where no more accurate method can be carried out, it is well to adopt the simple plan of allowing the milk to stand in a jug for an hour or more, and then, with as little shaking as possible, pour off the top half to be used in preparing the infant's food; a valuable increase of fat is obtained in this way.

Where even this is impossible the milk should be given with the least possible dilution consistent with the digestion of the casein, and, if necessary, the deficiency of fat must be made good by the daily administration of small doses of cod-liver oil. The writer has sometimes used a little butter, a piece about the size of a large pea, dissolved and shaken up well, in a three or four-ounce feed of diluted milk; the proportion of fat may be raised in this way nearly 1 per cent. Infants generally tolerate this amount of butter well, but the method is not to be recommended where cream can be obtained, for the butter does not mix easily with the milk, it has a taste which may be disliked, and it is often adulterated, especially with preservatives which may do harm.

Addition of Sugar.—Whatever strength of cow's milk is used the addition of sugar is necessary to bring the proportion up to that found in human milk. For this purpose milk-sugar is preferable to cane-sugar. A standardised solution of milk-sugar is sometimes used for the dilution of milk, but this has the disadvantage that the solution must be frequently made afresh to prevent its undergoing fermentation. It is quite as simple and usually much more convenient to add the milk-sugar directly to the diluted milk; and if it be remembered that one level teaspoonful (75 grains) of the powdered milk-sugar in a three-ounce mixture gives a proportion of about 5 per cent of sugar the calculation is simple enough. Thus in a three-ounce mixture of equal parts of milk and water, which, as already pointed out, contains 2 per cent of sugar, the addition of one level teaspoonful of milk-sugar will raise the proportion to 7 per cent; and in 6 oz. of such a mixture the addition of two teaspoonfuls of milk-sugar will have the same effect.

The use of milk-sugar is not a matter of great importance, cane-sugar will serve the purpose almost equally well, and amongst the poorer classes it is commonly used on account of its cheapness. If ordinary white lump sugar is used a lump about half an inch square should be used to a three-ounce mixture of milk and water.

Milk Mixtures.—Of recent years there is a growing tendency to the use of carefully calculated formulæ in the modification of milk for infant feeding, with the object of approximating more nearly to the standard of human milk. This, undoubtedly, is a step in the right direction, and it is much to be desired that such careful modification should be more generally carried out; but at the same time it must be remembered that an infant is not a test-tube, and that no amount of mathematical precision in the calculation of formulæ will eliminate the idiosyncrasies of the infant. We may determine to a nicety the percentage of each constituent in a mixture, but the much more serious difficulty still remains to know what particular percentage will suit any given case. There is no golden rule whereby to determine this; one can judge to some extent from the age of the infant, but the requirements of each individual case must remain to a large extent a matter of experiment. The proportions of fat and sugar which are required vary very little at any period during the first year of life; the former should be 3-4 per cent, the lower proportion being given during the first few weeks, the sugar should be 6-7 per cent. The proportion of proteid is the chief difficulty; the following proportions can only be taken as a rough guide, for infants of the same age differ considerably in their power of digesting the proteids of cow's milk:—

Age.	Percentage of Proteid.
First week	1 per cent or less
2nd week to 6th week	1·5 per cent
6th week to 3rd month	1·75 ,, ,,
3rd month to 6th month	2·0 ,, ,,
6th ,, ,, 8th ,,	2·5 ,, ,,
8th ,, ,, 12th ,,	3·0 ,, ,,

These proportions can be approximately obtained by diluting the milk according to the following table:—

	First wk.	2nd to 6th wk.	6th wk. to 3rd mth.	3rd to 6th mth.	6th to 8th mth.	8th to 12th mth.
Milk	1	1	2	1	2	3
Water	3	2	3	1	1	1

If the milk is diluted in this way, and the quantity of each feed is regulated according to the table given below, the addition of one teaspoonful of 48 per cent centrifugalised cream, and of milk-sugar in the proportion of about one teaspoonful to every 3 oz. of the mixture, will give satisfactory proportions. For example:—

	Formula.	Approximate Proportion.
First week	Milk, 1 tablespoonful	Proteid, 1 per cent
	Water, 2 tablespoonfuls	Fat, 3·8 ,, ,,
	Lime-water, 1 tablespoonful	Sugar, 6·5 ,, ,,
	Cream (48 p.c.), 1 teaspoonful	
	Milk-sugar, half a teaspoonful	

(Half this quantity to be given as a feed.)

	Formula.	Approximate Proportion.
2nd to 6th week	Milk, 2 tablespoonfuls	Proteid, 1·3 per cent
	Water, 3 "	Fat, 3·2 " "
	Lime-water, 1 tablespoonful	Sugar, 6·3 " "
	Cream (48 p.c.), 1 teaspoonful	
	Milk-sugar, 1 level "	
6th wk. to 3rd mth.	Milk, 3 tablespoonfuls	Proteid, 1·6 per cent
	Water, 3½ "	Fat, 3·2 " "
	Lime-water, 1 tablespoonful	Sugar, 6·5 " "
	Cream (48 p.c.), 1 teaspoonful	
	Milk-sugar, 1 level "	
3rd to 6th month	Milk, 4 tablespoonfuls	Proteid, 2·0 per cent
	Water, 3 "	Fat, 3·25 " "
	Lime-water, 1 tablespoonful	Sugar, 6·5 " "
	Cream (48 p.c.), 1 teaspoonful	
	Milk-sugar, 1 level "	
6th to 8th month	Milk, 8 tablespoonfuls	Proteid, 2·6 per cent
	Water, 2 "	Fat, 3·4 " "
	Lime-water, 3 tablespoonfuls	Sugar, 7·0 " "
	Cream (48 p.c.), 1 teaspoonful	
	Milk-sugar, 1½ "	
8th to 12th month	Milk, 12 tablespoonfuls	Proteid, 3 per cent
	Water, 2 "	Fat, 3·4 " "
	Lime-water, 2 tablespoonfuls	Sugar, 6·2 " "
	Cream (48 p.c.), 1 teaspoonful	
	Milk-sugar, 2 "	

If a weaker cream is used, so that larger quantities are required, it becomes necessary to take into account the proteid and sugar as well as the fat in the cream; and often it is convenient to use such a thin cream instead of milk as the basis of the mixture. The proteids and sugar in any cream are practically in the same proportion as in milk. An 8 per cent cream can be obtained as described above by taking the upper 10 ounces from a quart of milk which has been allowed to stand for three hours. Such a cream only requires dilution and the addition of sugar. For an infant between the ages of 6 weeks and 3 months such a mixture as the following is suitable:—

Formula.	Approximate Proportion.
Cream (8 p.c.), 3 tablespoonfuls	Proteid, 1·7 per cent
Water, 3 tablespoonfuls	Fat, 3·3 " "
Lime-water, 1 tablespoonful	Sugar, 6·6 " "
Milk-sugar, 1 teaspoonful	

At six months it may be used with an equal quantity of diluent, thus:—

Formula.	Approximate Proportion.
Cream (8 p.c.), 6 tablespoonfuls	Proteid, 2 per cent
Water, 5 tablespoonfuls	Fat, 4 " "
Lime-water, 1 tablespoonful	Sugar, 7 " "
Milk-sugar, 2 teaspoonfuls	

Similarly by using the upper 15 ounces (6·5 per cent fat) from the quart of milk, and diluting with an equal quantity of water, a mixture is obtained containing proteids 2 per cent, fat 3·25 per cent; or, by using the upper 5 ounces (11 per cent fat), and diluting with twice the quantity of water, a mixture is obtained with proteids 1·3 per cent, fat 3·7 per cent.

The percentages obtained by any such methods, even with the utmost care, are necessarily only approximate, for the composition of milk, even from the same cow, varies not merely from day to day, but even at different hours of the day; and although this variation is somewhat less in the mixed milk of commerce, it is still present, and in cream it is still more marked.

Some of the large London dairy companies now undertake the preparation of milk mixtures according to prescriptions from medical men, and this at once simplifies the matter; but the costliness of such a luxury places it at present beyond the reach of any but the wealthier classes. Home modification is much less expensive, and is quite satisfactory where proper care is taken. For the hard-working poor even such modification may be impossible, and for them any elaborate formulæ are mere counsels of perfection. Simple dilution with plain water, or barley water, according to the table given above, with the addition of a small lump of white sugar to each feed, often gives very good results; and if the deficiency of fat be made good by daily administration of cod-liver oil, infants fed thus may do perfectly well.

Quantity and Frequency of Meals.—Whatever food is used it is most important that feeding should be regular. The tendency is to feed an infant too often and with too large quantities. The infant who is fed "whenever it cries" cannot be expected to digest its food, and it is not surprising if an infant vomits when it is allowed to take a much larger quantity than the stomach can possibly hold without considerable over-distension. The average capacity of the stomach, according to Dr. Holt, is at birth 1⅕ oz., at three months 4½ oz., at six months 6 oz., and at twelve months 9 oz. Some post-mortem observations made by the writer agreed fairly closely with these measurements, and clinical experience has shown that at these ages these are about the quantities which a healthy infant is able to digest. Of course some latitude must be allowed for the constitution of the particular infant: a vigorous baby above the average size may be able to digest and may require a larger feed than a smaller infant of the same age. Some infants are habitually "small feeders," and will thrive on smaller quantities than those mentioned.

The following table may serve as a guide for the feeding of an average healthy infant:—

Age.	Interval between Feeds.	Quantity.	Number of Meals.	Total Amount in 24 hours.
First week . . .	2 hours	1 oz.	10	10 oz.
2nd wk. to 6th wk. .	2 "	1½ to 2 oz.	8	12 to 16 oz.
6th wk. to 3 mos. .	2½ "	3 to 4 oz.	6	18 to 24 oz.
3 mos. to 6 mos. . .	3 "	4 to 6 oz.	6	24 to 36 oz.
6 mos. to 8 mos. . .	3 "	6 to 7 oz.	6	36 to 42 oz.
8 mos. to 12 mos. . .	3 "	7 to 8 oz.	5	35 to 40 oz. (with additional food)

The infant should be accustomed, as in breast-feeding, to sleep several hours at night without being fed; the interval should be increased gradually from four hours during the first week up to six hours at three months, and with some infants seven or eight hours at six months.

Boiling, Pasteurisation, and Sterilisation of Milk.—Cow's milk and cream as they reach the consumer contain a considerable number of bacteria, and it has been proved beyond dispute that some of the acute specific fevers, particularly typhoid, scarlet, and diphtheria, have occasionally been conveyed by milk; recently, also, much attention has been paid to the transmission of tuberculosis in this way, and it has been shown that milk from cows with tuberculosis of the udder frequently contains the tubercle bacillus; the risk of infection is undoubtedly a real one, but the frequency of its occurrence has probably been greatly exaggerated. A much commoner and more widely disastrous infection from milk is that which produces summer diarrhœa; whatever may be the micro-organism which produces it, there can be little doubt that milk infection is chiefly responsible for the terrible mortality from infantile diarrhœa. (See "Gastro-intestinal Disorders of Infancy.")

For the prevention of these various risks three methods of preparing the milk are available: (1) Boiling, (2) Pasteurisation, (3) Sterilisation.

Boiling is at once the simplest and the least objectionable method of preparing the milk; but it must be understood that by "boiling" the writer means simply heating the milk just to the boiling-point, and then at once stopping the process. It is advisable to stir the milk frequently during the heating, so that the whole of it may be heated to the boiling-point. Bacteriological observations have shown that for all practical purposes milk which has been treated thus is a perfectly safe food so far as any risk of infection is concerned; even virulent tuberculous milk was found to be innocuous after exposure to a temperature of only 185° F. for five minutes (Woodhead). Prolonged boiling for five, ten, or fifteen minutes, and *a fortiori* for such periods as half an hour or one hour, is not only unnecessary, but in some way seems to impair the nutritive value of the milk. A sufficient quantity of the food to last twelve hours can be prepared twice daily; the milk and cream should be mixed before the boiling is carried out, but if lime-water is to be used it should be added subsequently, as some chemical change seems to be produced by boiling it with the milk.

After boiling, the mixture should be put at once into clean bottles, which are closed with tight-fitting stoppers, and should be rapidly cooled in cold water, or on ice, and then kept in some cool place until required.

Compared with pasteurisation boiling has the disadvantage that it gives the milk a taste which some infants may dislike, but, as a matter of fact, very few infants object to it, so that this is seldom a practical difficulty. The coagulation of the lactalbumin by boiling is also a disadvantage, as it reduces to some extent the nutritive value of the milk.

Prolonged boiling, like other methods of complete sterilisation, has been shown to produce some change in the milk whereby it loses its antiscorbutic power; and the continuous use of such milk is liable to produce infantile scurvy. So far all are agreed; but there is more to be said on this point. Infants come under notice every now and then who have been fed continuously on milk which has been boiled for fifteen minutes, or perhaps longer; they show no evidence whatever of pronounced scurvy, but they are "not getting on," they are peevish and miserable, their skin, instead of showing a clear healthy colour, shows an earthy pallor, only less marked than the anæmia of scurvy, their muscles are flabby, and they are not gaining weight as they should; there is, in fact, a cachexia, which is hardly defined enough to be called scurvy, but may well be the vanishing point of scurvy —and this appears to be due in some cases to overboiled milk, but much more often to sterilisation. If, however, milk be heated only just to the boiling-point, and then immediately cooled, it would seem that the risk of any such cachexia is considerably diminished.

Pasteurisation of milk consists in keeping it at a temperature of 155°-165° F. for twenty to thirty minutes. This is found to render the milk innocuous, so far as pathogenic micro-organisms are concerned, but like heating just to the boiling-point, or even boiling for two or three minutes, it does not necessarily render the milk "sterile" in the strict sense of the word.

Various forms of apparatus are made for this purpose under the name of "sterilisers," this term being loosely used to include both pasteurising and sterilising apparatus. The principle is the same in most of these forms: the milk is placed in an inner vessel which is surrounded by a jacket of cold water, into which a thermometer passes, the water is slowly raised to a temperature of 155°-165° F., and the source of heat is then removed; the milk is left surrounded by this heated water for twenty minutes. An ordinary deep tin saucepan, with a wire or perforated tin tray suspended in it, as in a fish-kettle, so that the bottle of milk may not be overheated by contact with the bottom of the saucepan, will suit the purpose admirably; a hole is bored in the lid, and a high temperature thermometer, which can be bought from any scientific instrument maker, passes through a cork fitted into this hole, into the water inside the saucepan. A glass bottle which contains the milk, and is stoppered with a plug of cotton wool, is placed on the tray in the saucepan, and surrounded with cold water up to the level of the milk; the saucepan is then placed on the fire, or more conveniently over a gas-jet, and the contents are slowly heated until the thermometer projecting through the lid registers 160° F., the saucepan is then removed from the fire, and after twenty minutes the bottle of milk is taken out, the cotton wool is replaced by a tight-fitting stopper, and the bottle is then rapidly cooled, and kept in some cool place until required for use.

Whether pasteurisation is altogether free from the risk of producing the cachectic condition which may result from prolonged feeding with sterilised milk, and, to a less degree, from the use of overboiled milk, is at least open to doubt.

Sterilisation means, properly speaking, the destruction of all bacteria and spores, so that milk thus treated will keep indefinitely. This is accomplished by subjecting the milk to prolonged boiling in the usual way for an hour or more, or by the use of superheated steam. For domestic purposes an apparatus exactly similar to that used for pasteurising is most convenient; the temperature, however, is raised to 212° for not less than forty minutes. Although in most cases this will render the milk completely sterile, it does not necessarily do so, the spores of some micro-organisms resist such a temperature for a much longer time.

Such a method of preservation is now widely practised for commercial purposes; sterilised milk can be obtained in most towns, and it is to be remembered that "humanised milk," as sold in the shops, is also sterilised. These sterilised preparations are very useful where, for any reason, for example in travelling, it is impossible to obtain fresh milk. In some cases, also, sterilised milk seems to be more easily digested than milk prepared in other ways, and some observers have obtained good results by feeding infants on undiluted milk which has been heated to 212° F. for forty minutes.

But what has been said of overboiled milk applies with much greater force to milk sterilised by prolonged exposure to a high temperature. The writer's experience has convinced him that the prolonged use of sterilised milk is responsible, not only for that comparatively rare condition which we recognise as fully-developed scurvy, but also for the less rare but not less important condition of malnutrition and cachexia which has been described above. In the present state of our knowledge it is impossible to explain why only a limited number of the cases fed thus develop these untoward symptoms, but it is very important that their relation to such feeding should be recognised.

To sum up, one may say that either heating just to the boiling-point, or pasteurisation, is much to be preferred to sterilisation, using this term in its strict sense. All these methods of preparation are to be regarded as necessary evils, not as virtues in themselves. The ideal to be aimed at is not the destruction of micro-organisms by processes which may, and almost certainly do, diminish the nutritive value of the milk, but the prevention of such contamination by properly regulated sanitation in cow-sheds and in the purveying of milk. Lastly, it may be pointed out that no method of preparation can make a bad milk good; a milk which is "only slightly turned" remains unfit for infant food whatever may be done to it, and a milk which is poor in fat does not become any the richer by being "sterilised."

Subsequent Care of the Milk.—Whatever precautions are taken in preparing the milk, it must be remembered that if exposed to the air afterwards it quickly becomes contaminated again with micro-organisms, and decomposes quite as rapidly, if not more rapidly than fresh milk. The jug in which it is placed should be cleansed with boiling water just before and after use, and, so long as the milk is in it, should be kept covered in a cool place. A better way is to use bottles properly cleansed with boiling water, which can be stoppered with sterilised wool, or with some tight-fitting stopper. In many of the special forms of apparatus now sold for pasteurising or sterilising, the milk to be prepared is already placed in feeding-bottles stoppered with wool or special stoppers, so that it is only necessary to replace the stopper by a teat when the infant is to be fed.

Feeding-Bottles and their Use.—The best form of bottle is the boat-shaped feeder, to one end of which a teat is directly attached. A rubber tube is to be avoided, it is almost impossible to clean it thoroughly, it becomes coated with stale milk and bacteria, and is a fruitful source of danger. Before and after use the bottle should always be well washed with boiling water, and in the intervals between feeds both bottle and teat should be kept in water to which a good pinch of borax has been added. The teat especially must be carefully cleansed, the inside is apt to become foul. Recently a mere conical cap which can be turned inside out for cleansing purposes has come into use, but it has the practical disadvantage that it lacks a shield, and a vigorous baby will sometimes nearly choke itself by cramming the whole of the teat into its mouth.

The size of the holes in the teat is a matter of some importance. Vomiting is sometimes due to the use of a teat in which the holes are so large that the child gets its food too quickly. On the other hand, the writer was recently consulted by an anxious mother on account of the supposed sudden failure of appetite in her infant. After sucking at the bottle a few minutes it would cry fretfully and refuse it. Examination of the teat, which was a new one, showed that the holes were so small that it was only just possible by very forcible sucking to get any milk through. Enlargement of these with a pin speedily set matters right. It makes little difference whether the holes are simple round punctures or of leech-bite shape so long as the child can draw the milk at a suitable rate.

The milk should be warmed to about 100° F. just before the meal; this may be done by standing the bottle, up to the level of the surface of the milk, in a jar of water as hot as the hand can comfortably bear for about five minutes, or more accurately by such an apparatus as has been described above for pasteurising, the thermometer being kept at 100° F. for about three minutes.

The bottle must be gradually tilted by the nurse as the child empties it, so that the nipple end shall be kept full to prevent the child sucking in air. The infant should be accustomed to take its food without interruption, the whole meal lasting ten to twenty minutes, after which any remaining milk must be thrown away, and the bottle washed and put away. The infant should not be allowed to take its food in a desultory way, lingering over its bottle for half an hour or more, or still worse, being allowed to suck even after the bottle is empty in order to keep it quiet.

Additional Food.—Supposing an infant to be making satisfactory progress on diluted cow's milk, the question arises, How soon should any additional food be given? As a general rule no other food of any kind is required until the infant is at least seven months old, and many infants will do well without any addition to their diet until nine months old. The best guide is the weight of the infant, so long as this is increasing satisfactorily the diet is sufficient.

As early as the third or fourth month a small quantity of a completely malted food, such as Mellin's, can be added to one or two of the feeds daily without doing any harm, but it is seldom necessary. At the age of six or seven months one of the partially malted foods, such as Allenbury No. 3, or Nestlé's Food, which contain some unconverted starch, may be cautiously added to one or two meals in the day. If these are well taken, some prepared wheat flour or a Robb's biscuit may be given once or twice a day at the age of nine or ten months, and about the same time the yolk of an egg lightly boiled may be given occasionally. At the age of twelve months a tablespoonful of crumbled stale bread or a rusk soaked in milk or in red gravy from underdone meat may be allowed once a day, and a little later mealy potato mashed into a fine flour and beaten up with milk may be given two or three times a week.

Milk of Animals other than the Cow

Asses' Milk, Goats' Milk.—Unfortunately there are some infants who will not thrive on cow's milk however carefully it is diluted, and for such it is necessary to find some other method of feeding.

For those who are able to afford it the milk of the ass or the goat is sometimes successful as a substitute for mother's milk, but by no means always, and such milk should never be ordered without due regard to the cause of the failure to thrive on cow's milk. This will be better understood from a comparison of the analyses of these various milks:—

	Human Milk.	Cow's Milk.	Asses' Milk (Winter-Blyth).	Goat's Milk (Winter-Blyth).
Proteid .	2·0 { Casein, ·6 Lact-albumin, 1·4	4·0 { Casein, 3·25 Lact-albumin, ·75	1·8 { Casein, 1·0 Lact-albumin, ·8	3·7 { Casein, 3·0 Lact-albumin, ·7
Fat . . .	3·5	3·5	1·0	4·2
Sugar . .	7·0	4·0	5·5	4·0
Salts . .	·2	·7	·4	·5

(Under the head of lactalbumin in the analyses of asses' milk and goat's milk peptones amounting to about ·1 per cent have been included for convenience.)

It will be seen that asses' milk is much poorer than cow's milk both in proteids and in fat (in a specimen examined by the writer the fat amounted to only ·3 per cent). The casein forms a very fine flocculent curd almost like that in human milk, and therefore may be digested well, where, as usually happens, the trouble in digesting cow's milk is due to the casein. Its extreme poorness in fat, however, makes it unsuitable for continued use, and asses' milk must be regarded only as a temporary food suitable for very young or very weakly infants, or for such a feeble condition of digestion as is left after a severe attack of gastro-enteritis. It has a laxative action, which is also sometimes useful, but may make it unsuitable where there is any tendency to diarrhœa.

Goat's milk, on the contrary, is almost as rich in casein as cow's milk and is richer in cream. It might be expected, therefore, that it would be useless ordering goat's milk where there is difficulty in digesting casein; this, however, does not necessarily follow, for in some observations made by the writer, the curd given by goat's milk with hydrochloric acid was much more finely divided than that given by cow's milk under similar conditions. A trial of goat's milk may therefore be useful even where the infant is unable to digest the casein of cow's milk. Some infants thrive excellently on goat's milk, and in the country where a field is available it is not an expensive matter to keep a goat. One infant under the writer's care was fed on goat's milk for several months; the goat cost fourteen pence a week to keep, and yielded at least two to three pints of milk twice a day. Goat's milk, of course, requires dilution in the same way as cow's milk.

Ewe's Milk and Mare's Milk have also been used for infants. The former is even richer in fat and casein than goat's milk, and is hardly likely to suit an infant who cannot digest cow's milk. Mare's milk is slightly poorer in fat and casein than cow's milk, and occupies in this respect an intermediate position between cow's milk and asses' milk.

With the more intelligent modification of cow's milk which is practised nowadays, the necessity for the use of the milk of other animals has become less, and it is now comparatively seldom that any of those mentioned here are used.

MILK PREPARATIONS

"*Humanised Milk,*" as sold by many of the larger dairy companies, is of considerable value as a temporary food, where for any reason it is impossible to carry out the careful modification of cow's milk at home.

The term "humanised" simply means that the percentages of fat, proteid, and sugar have been modified to bring the milk as near as possible in composition to human milk, and the principles of preparation are exactly the same as those already described for the home modification of milk. Some humanised milk, however, has its digestibility further increased by partial pancreatisation, a process which will be described below.

Humanised milk is now commonly sold in two or three different strengths, so that the percentage of proteid can be increased as the infant's powers of digestion increase.

As a temporary measure such milk is often of great value, and the writer has obtained excellent results from it; but it is apt to be forgotten that these commercial preparations have been sterilised, usually by prolonged exposure to high temperature, and have not therefore the full value of fresh milk. In this way they are inferior to the home modifications of fresh milk. It happens not uncommonly that an infant thrives well for a few weeks, or two or three months, on "humanised milk," and then begins to fail and to show the same vague symptoms which have already been described as due to the prolonged boiling or sterilising of milk. These sterilised preparations, therefore, should not be used alone for long periods; it is preferable, if possible, to modify fresh milk at home as directed above.

Peptonised Milk.—Under the name of peptonisation are generally included two different processes for the predigestion of foods. In the one the ferment of the gastric juice, pepsin, is used; in the other the ferments of the pancreas. The latter is by far the more convenient to use for the predigestion of milk, for not only is it said to act with special ease on the casein of cow's milk, but it also acts in an alkaline or neutral medium, whereas pepsin requires a distinctly acid medium. By this predigestion the proteids including the casein are more or less completely converted into soluble peptones which are easily absorbed.

Various methods of peptonisation are in vogue. A very simple and effectual method is to mix a quarter of a pint of boiling water with half a pint of cold milk, then add the following powder: Extract. pancreatis (Armour's) gr. ijs., sod. bicarb. gr. x., and allow to stand in front of the fire for ten minutes with occasional stirring, then heat the mixture just to the boiling-point. This final heating stops the action of the ferment, which if continued too long gives an unpleasant taste to the milk; the heat at the same time serves to destroy any micro-organisms in the milk. Fairchild's peptogenic milk powder is very similar in composition, and used according to the directions supplied with it produces an easily assimilated milk mixture, in which the casein is partially digested, and which, according to Dr. Leeds's analysis, closely resembles human milk in its composition. Benger's liquor pancreaticus is used thus: Half a pint of boiling water is mixed with a pint of cold milk, two teaspoonfuls of the liquor with 20 grains of sodium bicarbonate are added, and the whole mixture is kept in front of a fire for about one hour and then boiled. If used without further dilution this mixture requires the addition of about half a teaspoonful of milk-sugar to every three ounces. Fairchild's zymine (gr. v. with sod. bicarb. gr. xv., the contents of one of the glass tubes in which it is sold) can be used similarly.

Whatever method is adopted the degree of peptonisation can be regulated by the time the peptonising process is continued. In discontinuing the use of peptonised milk it is often useful to reduce the time of peptonisation gradually, so that more and more of the work of digestion may be left to the infant's stomach. In this way many an infant who seems unable to digest cow's milk may gradually be coaxed into digesting it without artificial aid.

For some conditions peptonised milk is particularly valuable. The miserable emaciated infant, who, in spite of much care in the dilution of milk, is continually whining or screaming with flatulence and colic, whose bowels are costive, and stools consist largely of undigested curd, will often turn the corner and make rapid progress on peptonised milk; so, too, the infant who is just convalescent from an attack of gastro-enteritis, and whose stomach, as so often happens, seems to have lost in great measure its digestive power, is often benefited by the use of peptonised milk for two or three weeks.

But there are certain disadvantages attaching to peptonised milk which it is well to remember. It has a laxative effect which is not always desirable, and in some infants causes troublesome diarrhœa. It must, therefore, be used with caution where there is any tendency to looseness of the bowels. If this predigested milk is used too long it is sometimes very difficult to return to ordinary milk, the stomach refuses to do its work after having it done for it so long. Lastly, in the process of peptonisation the milk loses its antiscorbutic value, and an infant fed for several weeks on peptonised milk may develop infantile scurvy.

Whey is often very useful for infants who are unable to digest casein. It is made either by the addition of an acid or by the action of rennet on milk. A convenient method is to use essence of rennet or Benger's curdling fluid. Add one teaspoonful of either of these to half a pint of milk, which has been warmed to about 98° F.; let it stand before the fire until the curd has set, then break up the curd thoroughly with a fork, and allow it to stand for fifteen minutes, then strain off the curd, and heat the whey to the boiling-point.

Where rennet or essence of rennet is not obtainable, lemon juice may be used in the proportion of one teaspoonful to half a pint of milk; such lemon whey is particularly useful as a food in infantile scurvy. In some cases sherry whey is more useful, especially where it is desired to combine a stimulant with the food; in the extreme exhaustion of acute gastro-enteritis, or in the late stages of marasmus, few foods are so well taken. For infants, also, whose digestion has become so enfeebled by a long course of unsuitable diet that they seem scarcely able to digest anything, the sherry whey seems to act both as a stimulant to the gastric function and an easily assimilated food, so that, after a few days, it is possible to add cautiously other food, and so gradually to encourage the infant to take a more nourishing diet. Sherry whey is made by heating half a pint of milk just to the boiling-point, adding a good wineglassful of sherry, then heating to the boiling-point and allowing to stand until the curd has settled; the curd is then separated from the whey by straining through muslin.

Whey is milk from which the casein has been removed; but the casein in its precipitation carries with it the greater part of the fat globules, so that the constituents of whey are the lactalbumin, sugar, and salts of milk, with very little of the fat and practically none of the casein. It is, therefore, not sufficiently nourishing for prolonged use; the proportion of fat, in particular, is seriously deficient; it amounts, according to Koenig, only to ·24 per cent, while the proteid amounts only to ·82 per cent. If whey is to be used for more than a few days, therefore, it is generally advisable to add cream; and many infants will make excellent progress for several weeks on a mixture of whey and cream alone. The addition of a little raw meat juice to two or three feeds, or the substitution of one or two feeds of veal broth or chicken broth, will rectify the low percentage of proteids. A useful mixture may be obtained by adding cream and sugar in the following proportions:—

Whey	4 tablespoonfuls.
Cream (48 per cent)	1 teaspoonful.
Milk-sugar	Half a teaspoonful.

For an infant who is able to digest more proteid a small quantity of casein may be added by using the weaker home-made 8 per cent cream; thus,

Whey	6 tablespoonfuls.
Cream (8 per cent)	2 „
Milk-sugar	1 teaspoonful.

When the digestion is still further improved, ordinary milk may be added to the whey (after this has been boiled to stop the action of the curdling ferment), and such a mixture as the following may be given:—

Milk	3 tablespoonfuls.
Whey	3 „
Cream (48 per cent)	1 teaspoonful.
Milk-sugar	1 „

Where cream or milk is used with whey it is necessary to use rennet-made whey which has been boiled—not the acid or sherry whey, for in these the acidity may curdle the added cream or milk.

Koumiss may be mentioned here, but is rather suitable for children over the age of two years than for infants. As originally used by the Tartars, it was made from mare's milk or asses' milk, with kefir grains, consisting of yeast and bacteria. In this country it is made from cow's milk with ordinary brewers' yeast.

At the Hospital for Sick Children, Great Ormond Street, a not unpleasant preparation has been made by the following method:—Mix together new milk 40 oz., water 40 oz., brewers' yeast 1 oz., and loaf sugar 3 oz., in an open jar; allow the mixture to stand at 30° C. for five or six hours. When bubbles of gas begin to rise in it, add milk 40 oz., water 40 oz., and milk-sugar 4½ oz.; then bottle, being careful to secure the corks with string or wire. Keep in a cool place for five days before using. It must be tapped with a champagne cannula, not by removing the cork. This has been used with satisfactory results for older children in convalescence from acute disease, and is sometimes useful for infants with troublesome vomiting. It contains alcohol in very small quantity, 1-2 per cent, about a quarter of the amount present in ordinary London porter, and therefore is to some extent a stimulant. It has a somewhat beery taste, and is markedly acid. It is very deficient in fat, and in this respect is a poor food for young children, except for very short periods. Prepared according to the method described above, it contained, in two samples examined by the writer, 1 per cent of fat, and about the same proportion was found in samples obtained from shops, but one supplied by a London dairy company contained only ·05 per cent of fat.

In these days of sterilisation, it is interesting to note that the value of koumiss as a food in certain cases is undeniable; but none the less bacteriological examination showed in all the specimens examined by the writer that it was swarming with micrococci, as appears to be always the case from its method of preparation.

Condensed Milk.—This is cow's milk which has been evaporated *in vacuo*, and then sterilised by heat. It is sold in two varieties—(1) sweetened, (2) unsweetened. The better class brands are made from whole milk, *i.e.* milk from which none of the cream has been removed, or even from milk to which cream has been added; but a very large quantity of the cheap condensed milk is made from skimmed or separated milk which has lost nearly all its cream, and which, therefore, even if it were fresh, would be totally unfit for infant food.

The ordinary sweetened condensed milk contains a large quantity of added cane-sugar. The average composition of the better class brands, as calculated from published analyses quoted by Blyth, is as follows:—

Average Sweetened Condensed Milk.

Proteids	10·9 per cent	
Fat	9·5 „ „	
Sugar	54·2 „ „	Cane-sugar, 41·1 Milk-sugar, 13·1
Salts	2·03 „ „	
Water	21·37 „ „	

It will be seen that in order to reduce the percentage of sugar to the correct proportion, about 7 per cent, it is necessary to dilute nearly seven times, with the following approximate result:—

Average Sweetened Condensed Milk diluted with Seven Times its Quantity of Water.

Proteids	1·3 per cent
Fat	1·1 „ „
Sugar	6·7 „ „
Salts	·2 „ „
Water	90·7 „ „

Such a mixture contains altogether too little fat, but the directions on some of the brands recommend even further dilution. For instance, in one (Sledge Brand) examined by the writer, the fat amounted to 9·5 per cent, the directions recommended dilution with 11 parts of water for the first month, and then with 8 parts of water for the second and third months, making the proportions of fat respectively ·8 per cent and ·1 per cent. The proteids, also, with such dilution, become very deficient. As a matter of fact these directions are rarely carried out with any accuracy, for the milk is measured with a teaspoon, which is then used to stir it into the water; condensed milk, being very thick, clings to the teaspoon, which may thus contain as much as 3 or 4 drachms. In this way the writer has found the percentage of fat, where condensed milk was prepared nominally in the proportion of a teaspoonful to 4 ounces of water, to be as much as 3·2 per cent. In such a case, however, the lessened dilution of fat meant also lessened dilution of sugar, which was altogether in excess (probably about 18 per cent). There is, in fact, no *via media* in using the ordinary sweetened condensed milk, either the sugar must be seriously in excess, or the fat must be seriously deficient.

With the inferior sweetened condensed milk which the poor buy at about half the price of the better class brands, the deficiency of fat is even worse. The proportion of fat in some of these was as low as 0·4 per cent (Dyer), and as the sugar is as excessive as in the better class brands they require similar dilution; the result is a proportion of fat, in some of these, amounting to barely ·05 per cent. It is hardly to be wondered at that rickets and marasmus should result from such a diet.

Unsweetened condensed milk of a reliable brand is much more satisfactory in this respect; it contains no cane-sugar, and requires much less dilution, so that the percentage of fat is not so greatly diminished. In the manufacture of some of these brands the fat has been increased by the addition of cream before condensation. The Viking Brand, in the sample examined by the writer, contained 11 per cent of fat; the "Ideal" contained 10·4 per cent; the "First Swiss" (Romanshorn), 9·4 per cent. These require dilution with about three times their quantity of water to bring the other constituents to suitable proportions: the percentage of fat thus becomes 2·75, 2·6, and 2·35 respectively. Milk-sugar should be added to this unsweetened milk in the proportion of half a teaspoonful to 3 ounces of the diluted mixture. The unsweetened milk should be used within thirty-six hours of opening the tin; it decomposes much more rapidly than the sweetened milk, in which the cane-sugar acts as a preservative. Condensed milk has its value as a temporary food, where it is impossible to obtain fresh cow's milk, or where an infant is unable to digest ordinary diluted cow's milk. With an acid condensed milk gives a large firm clot, very like that formed in fresh milk, but, in spite of this, it is certainly more easily digested by some infants. If its use has to be continued several weeks the deficiency of fat should be corrected by the addition of ½-1 teaspoonful of fresh (48 per cent) cream to each 3-4 oz. feed, so as to bring the fat in the mixture to at least 3 per cent. The unsweetened milk should always be used in preference to the sweetened; but if only the sweetened is available (the other costs rather more, owing to its more rapid decomposition after opening, and therefore cannot always be used by the poor), it should be diluted freely, so as to reduce the sugar to 6-7 per cent, and then enriched by the addition of cream. It may also be necessary to give raw meat juice once or twice a day to correct the deficiency of proteids.

Condensed milk should not be used as the only food for an infant, except for very short periods. A very large proportion of the infants fed entirely or almost entirely on condensed milk develop rickets, partly, no doubt, owing to the excess of sugar, but perhaps more on account of the deficiency of fat. Moreover, in the process of condensation the milk has lost its antiscorbutic properties, and the use of condensed milk is a not uncommon factor in the etiology of infantile scurvy.

PROPRIETARY FOODS

The name of these is legion, and it would be impossible to describe them all in detail. There are few of them which are not occasionally of use, and still fewer which could not be usefully dispensed with. Almost all of them are preparations of cereals, and as such, almost all contain starch, but with this important difference, that while in some of them the starch has been partially or completely converted into the soluble carbohydrates, dextrin, maltose, or glucose, in others the starch remains unaltered. It is upon the degree of this conversion of the starch and upon the amount of fat present that the value of any individual preparation chiefly depends. None of the so-called "infant foods" which contain unconverted starch should be given to an infant under the age of six months; most infants are much better without any of these preparations until the age of nine months, and even then only those in which the starch has been partially converted should be allowed.

A very serious defect in all these "foods" is the low proportion of fat; some, indeed, scarcely contain any fat at all, and, prepared as they commonly are by the simple addition of water, they are totally unfit for infant food. In some of them this deficiency is to some extent corrected by giving them with diluted milk; but the fat value of the mixture is scarcely greater than that of the diluted milk alone, so that the addition of cream would be necessary to make the fat sufficient.

The scurvy-producing tendency of all these feeds is now well recognised, and, indeed, in the majority of the cases of pronounced infantile scurvy which have come under the writer's observation, the infants had been fed on one or other of these so-called "infant foods." Why they should produce scurvy is less evident; but this much seems clear, that the fault is a negative one rather than a positive: it would seem that in some way the food is "devitalised" in the process of manufacture, as milk is by sterilising, and to a lesser degree by boiling, and that the lack of this "vital element," if one may use such a vague term, is the cause of the scurvy. Here, again, the writer would like to emphasise the fact that weeks, perhaps months, before an infant shows any definite symptoms of scurvy, there may be a condition of cachexia and malnutrition which is as definitely related to the use of these sterilised "infant foods" as is the classical condition with spongy gums and subperiosteal hæmorrhages. If, therefore, any of these preparations are to be used for more than a few weeks, an adequate supply of fresh food of some sort should be given; and it may be pointed out in this connection that the addition of only a small quantity of milk, especially if it has been boiled, is not necessarily sufficient to prevent the development even of severe scurvy. The antiscorbutic power of milk is definite, but feeble; and if only small quantities of milk can be given, it may be necessary to supplement it with raw meat juice or lemon whey.

Such methods, however, are but unsatisfactory at the best, and the need for them should not be allowed to arise. There are very few cases in which any of these proprietary foods should be allowed to become the principal article of diet; and, in spite of all the advertisements, there is not a single "infant food" in the market which can adequately replace cow's milk as a food for infants.

With all their very serious faults, however, some of the proprietary foods have their value.

Dried milk preparations in particular are often well digested by infants who are quite unable to digest ordinary cow's milk; some of the best known of these are the Allenbury foods, No. 1 and No. 2, and Horlick's malted milk. These contain no starch, and when diluted with water according to the manufacturer's directions they contain respectively 2·6 per cent, 2·4 per cent, and 1·6 per cent of fat (the latter was obtained when four teaspoonfuls of Horlick's malted milk were added to four ounces of water). The curd produced by the addition of an acid to these preparations is much finer than in ordinary cow's milk, and seems to be easily digested. Such preparations are useful in some cases of gastro-intestinal disorder in infancy, but should only be used as temporary foods.

Another milk preparation is Nestlé's food, but this differs from the preceding in containing a considerable quantity of unconverted starch, together with some soluble carbohydrates; it is, in fact, a combination of a milk food and a partially malted cereal food. When diluted according to the directions it is very deficient in fat, containing considerably less than 1 per cent, and the presence of starch makes it wholly unsuitable for infants under the age of six months; after that age it may be useful given once or twice a day to accustom the infant gradually to a starch-containing food.

Completely malted cereal foods can be given at a much earlier age than those which contain unconverted starch. Perhaps the best known example of such a preparation is Mellin's food. This contains no starch, and consists chiefly of soluble carbohydrates, together with a fair proportion of proteid. It contains only a trace of fat (·18 per cent), but as this food is always to be used with cow's milk the fat deficiency is less important. Such a preparation as this, which is intended as an addition to, not a substitute for, cow's milk, is much less open to objection than the other foods, and there can be no doubt that in some cases the addition of one teaspoonful or two of Mellin's food to one or two of the milk foods daily proves useful, not only by its own nutritive value, but also by facilitating the digestion of the milk. Mellin's food has, however, a laxative effect, which, although often useful, may contra-indicate its use where there is any looseness of the bowels. As already said, milk properly diluted is usually the only food an infant requires until the age of six or seven months, but where there is difficulty in digesting casein, especially if there is also troublesome constipation, and nutrition seems to be failing, a small quantity of Mellin's food may be given occasionally in the milk with advantage at as early an age as three months.

Partially malted cereal foods which contain some unconverted starch form the majority of the advertised "infants' foods." Such are the Allenbury No. 3 food and Savory & Moore's food. The latter, when mixed with a warm fluid, either water or milk, as directed, undergoes changes by which the starch is partially converted, owing to the presence of malt diastase in the dry preparation. Benger's food is somewhat similar, but the partial conversion of starch is accomplished by pancreatic ferments.

Such foods are very deficient in fat, and unless made with milk are wholly unsuitable for the feeding of infants; used with milk, however, they are sometimes of service during the later months of the first year. They should not be given with every feed, once or twice in the day is usually sufficient, starch being thus cautiously introduced into the diet.

Farinaceous foods, in which there has been little or no conversion of starch, such as Ridge's food, Neaves' food, Frame food, entire wheat flour, and the ordinary home-made arrowroot or corn-flour, and the various "biscuits," can seldom be taken with advantage earlier than the tenth month, and in most cases are better avoided until the end of the first year. They all have their use, and are valuable additions to diet in the second and later years. It is true that a certain number of infants can and do digest such foods as early as the seventh month, but these are the exceptions, and more often the result is flatulence and colic, if not more serious disturbance of digestion. The use of such foods during the first six months of life is a common cause of the pitiable condition of misery and marasmus which is so frequently seen amongst the infants of the poor.

The proportions of the more important constituents of some of the best known of the proprietary foods may be seen from the following table, which has been compiled from analyses published by various observers, several by Dr. Leeds:—

Composition of Proprietary Foods.

	Allenbury No. 1.	Allenbury No. 2.	Horlick's Malted Milk.	Mellin.	Nestlé's Food.	Savory & Moore.	Allenbury No. 3.	Ridge's.
Starch	0·0	0·0	0·0	0·0	36·86	36·36	60·01	77·96
Soluble carbohydrates (Sugar, dextrin, etc)	65·48	69·02	63·59	68·18	40·9	44·83	25·1	5·19
Fat	13·15	12·48	8·4	0·18	4·25	0·43	1·05	0·63
Nitrogenous bodies	14·25	12·5	21·85	10·07	11·0	9·63	10·23	9·24
Ash	4·75	4·08	3·95	3·75	1·7	0·89	0·6	0·6

EGGS AND MEAT FOODS

The value of eggs as a fat-containing food is perhaps hardly sufficiently appreciated. The yolk of an egg contains nearly 30 per cent of fat, and in this respect is a most desirable food for infants. As early as nine months of age an infant may have the yolk of an egg very lightly boiled once a day, and during the second year eggs are an important constituent in the dietary of infancy. The white of an egg is of value chiefly as a proteid-containing food, and is occasionally a useful addition to the diet where an infant is unable to digest the proteids of cow's milk, and is being fed with a mixture of cream and water, which, although containing a sufficiency of fat, contains very little proteid. The white of the egg must be given raw, and the most convenient way to give it is in the form of "albumin water."

The white of one raw egg is cut in several directions with a clean scissors and then mixed with half a pint of water, and flavoured with a little cinnamon water.

Given in this form the white of an egg is often very useful as the only food for twenty-four or forty-eight hours in cases of severe vomiting and diarrhœa.

Raw meat juice is used similarly to supply the proteid element where this cannot be taken in the form of cow's milk. Like the yolk of an egg, raw meat juice is rich also in iron and in phosphorus, and is specially valuable in rickets and anæmic conditions. An infant aged six months may take a teaspoonful of raw meat juice, either sweetened and given alone in a spoon, or mixed with the diluted milk (after it is cool), or with cream and water, three or four times a day: to an infant of twelve months, two tablespoonfuls of raw meat juice may be given in the twenty-four hours.

Raw meat juice is made thus: scrape a quarter of a pound of lean raw beef into shreds in a cup; add to this two tablespoonfuls of cold water, and leave it, covered with a lid, to stand in a cool place for one hour; then strain it, and squeeze the juice out of the raw beef through muslin.

Meat broths are seldom to be recommended for healthy infants under the age of nine months, but in cases of troublesome vomiting or diarrhœa they may be given at the age of six months, or even earlier, as a temporary food. There are infants who seem unable to tolerate fat for a time, but who will take well a diet consisting chiefly of proteids; in such cases veal broth or chicken broth may be given with occasional feeds of whey, to which a little milk or cream may be added gradually, and in this way a return to a proper proportion of fat may by degrees be accomplished. Mutton broth, or, still better, veal or chicken broth, are to be preferred to beef tea, even when this is made very weak, for the feeding of infants; and, in the writer's opinion, the concentrated meat preparations which are now so widely sold are better avoided in the feeding of healthy infants. In cases where only very small quantities of food can be retained, and the infant is becoming exhausted by severe vomiting, a few drops of some concentrated meat preparation are often valuable as a temporary food.

DIET FROM TWELVE TO EIGHTEEN MONTHS

At the end of the first year an infant should have five or six meals in the twenty-four hours, the first being at 7 A.M., the last at 9 or 10 P.M. Cow's milk must still be the chief article of diet; but two or three feeds in the day may now consist of such starch-containing foods as finely crumbled stale bread soaked in milk or gravy; or a sponge-cake or rusk may be given sopped in milk. Most infants at this age like crumbled bread soaked in the fat from fried bacon, and it is a useful addition to the diet at this age; a few months later, thin slices of bread soaked or fried in the fat may be given. The yolk of an egg lightly boiled, or a custard made with egg, should be given occasionally, and once a day a little meat broth may take the place of milk. There is no objection at this age to such foods as Chapman's entire wheat flour, or Frame food, or a little well-boiled porridge once a day, and a thin slice of bread and butter may be allowed at two of the meals.

DIET FROM EIGHTEEN MONTHS TO TWO YEARS

After the age of eighteen months most infants require only four feeds in the day. At least a pint of milk should be taken in the twenty-four hours, but, in addition to the food already allowed, other articles may be given. Frame food or well-boiled oatmeal, with a drink of milk, will make a suitable breakfast, and for one meal in the day there is no objection to a tablespoonful of well-cooked and finely-mashed potato, which may be mixed with milk, or with the red gravy from underdone meat. Undoubtedly some infants can take potatoes without harm even earlier than this, but a very large number, not only of infants, but also of older children, suffer from disorder of digestion in consequence of being allowed potato too early or in too large quantity. It is a food which most children like, and which parents are only too ready to give, but it is quite unnecessary, and most children are far better without it until they are at least eighteen months old. There are few foods which are more often badly cooked than potatoes, and even when they are properly cooked they require to be finely mashed, not merely pressed through a fork, which leaves blocks of unmashed potato to worry the infant's digestion.

A tablespoonful of meat, very finely minced, or torn into fine shreds, may be given occasionally with the potato; underdone roast beef or mutton, or the white meat from chicken or turkey, or a little boiled sole or turbot well pounded are most suitable, and the quantity may be increased gradually.

Milk puddings, rice, tapioca, sago, or semolina properly cooked may be given with a little juice from cooked fresh fruit or from prunes; junket or blanc-mange will also serve to vary the diet.

Bread and butter, rusks and sponge-cake, can be given more freely as the child approaches the end of the second year; but here a word of caution is necessary: whatever solid food is given, the child must be taught to eat slowly and masticate thoroughly; any one who has watched an infant cramming its food into its mouth, and bolting it with little or no attempt at mastication, can hardly wonder that indigestion is the result; nor is this fault limited to later infancy, older children are too often habitual food-bolters, and it may require patience and careful training to accustom them to take their food properly.

Lastly, it may not be out of place to mention the value of cold water. Many parents and nurses seem to be afraid to give an infant cold water to drink, as if it had some baneful effect; and, as a consequence, one sees, especially in hot weather, infants crying fretfully, not from hunger, but from thirst, and they are soothed at once by a drink of cold water. Pure cold water in small quantities—not, of course, to take the place of food, but to relieve thirst—may be given at any period of infancy; and nurses often need to be told that an infant who is ill, with a high temperature, should have the cold water that it craves.

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Infection.

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BY "infection" is understood the introduction into the body of a pathogenic micro-organism which is capable of multiplying within it. Diseases, therefore, which depend on such an infection are called "infective" or "infectious" diseases. For a long time it was the custom to draw distinctions between infection and contagion, but, broadly speaking, the process is the same in every case, and all so-called contagious diseases are certainly infectious in the modern sense of the term. It is true, of course, that certain infections are only contracted by direct contact, as for instance syphilis, rabies, and gonorrhœa. But others, such as anthrax and small-pox, can be contracted not only by direct contact, but also through the intermediary of the air, and are thus, to use the old terms, both contagious and infectious. It is more convenient, therefore, to class all diseases which depend on the entrance of a living organism into the body as "infectious."

The living organisms which cause these diseases are nearly all vegetable in their nature, and are of the class "fungi." The vast majority of them are bacteria. But besides these vegetable organisms certain protozoa may give rise to infectious diseases, the most notable instance being the spirillum of relapsing fever. This is not the place to discuss or open up the whole question of bacteriology. It is sufficient to note that bacteria are divided into two main groups, that of (*a*) parasites, which can exist and thrive in living animal tissues, and that of (*b*) saprophytes, which can live outside living animal tissues, on dead animal or vegetable matter, or on inorganic substances. The first group, again, may be subdivided into those germs which cannot exist outside living animal tissue, "obligatory parasites," and those which can do so for a longer or shorter period, "facultative saprophytes." And the same subdivision may be made of the second group into "obligatory saprophytes" and "facultative parasites."

Each micro-organism, whether it be a bacillus or some variety of coccus, has the power when growing on the living tissues or in suitable media of manufacturing a toxine which is peculiar to itself. In an infectious disease the majority of its distinguishing symptoms are due to the toxine rather than to the presence of the bacillus itself, although the latter may in certain diseases be the cause of local conditions which may ultimately cause the death of the infected person. How the toxine itself is produced opens up a question of the greatest obscurity. Some hold that it is directly derived from the cell plasma of the bacillus, and shares the specific qualities of that bacillus. Others, again, regard it as the result of the action of the bacillus on proteid substances.

The micro-organism is introduced into the human body in a variety of ways. It may be in the first place directly inoculated. It may be inhaled. It may be ingested. In order that it should be introduced in one of these ways, it must either contaminate various articles with which the person attacked is in contact, or else be carried in the air, or, lastly, contaminate food or water. The germs of some diseases, such as small-pox and measles, are probably in most cases inhaled; that of typhoid fever, on the other hand, is always ingested. It is possible, however, that germs which are primarily inhaled may find their way from the mouth and pharynx to the alimentary canal. This would give an easy explanation of those cases of typhoid which seem to be due to dust. The action of insects in assisting the spread of infection depends on their contaminating food. (See article "Disinfection," vol. iii.)

After obtaining an entrance to the body, the germ apparently lies more or less latent for a time before it gives off any marked amount of toxines. This latent period, the period of *incubation*, distinguishes an infection from an intoxication, no such latent interval occurring in the case of the latter. This period may be long or short. Various infectious diseases show a range from a few hours up to nearly four weeks, some indeed much longer. Moreover, the period of latency is by no means constant in the case of any given disease, the variation, for instance, in typhoid fever being nearly three weeks. It would appear that in some diseases, at least, the length of the incubation period may be affected by the dose of infection which the subject has undergone. The larger the dose the earlier the disease may begin.

It must always be remembered, in computing the length of an incubation period, that the reckoning must be made from the exposure to infection to the first rise of temperature. Needless confusion is sometimes caused by dating up to the time when the rash of an exanthematous fever appears. Again, it must be remembered in cases where the latent period has appeared unduly long that it is perfectly possible that infective matter may have been carried about on the clothes or elsewhere by the patient, and that the real date of infection was perhaps a week subsequent to the last exposure.

When an infection has occurred it may be local or general. In the first case, as for instance diphtheria or tetanus, the germs obtain a nidus at a given spot and cause some local disturbance and inflammation. They are not found in the blood, and the constitutional disturbance set up is due to their toxines and not to themselves. On the other hand, in a disease like anthrax the bacilli are found in the blood-vessels and the infection is general. Not only are the toxines in such cases given off probably more rapidly, but the mere mechanical presence of the bacilli may cause lesions of great importance, such as emboli.

Again, an infection may be specific or non-specific. In many diseases we know that the germ with which they are identified always causes an exactly similar series of symptoms and of pathological lesions. Such infections are truly specific. But there are other diseases which are due to micro-organisms, and so must be classed as infective, which present the same clinical and pathological features, and yet may be found in different instances to be due to perfectly distinct germs. Thus pneumonia may be due to more than one organism, and the same holds true of ulcerative endocarditis. Suppuration, again, may be caused by quite a number of different micro-organisms. (See "Micro-organisms" and "Suppuration.")

The exact progress of an infection when once the germs have obtained an entrance into the body remains a question for speculation. No doubt a certain number of them are destroyed by the phagocyte cells, but these cells are probably without action on the toxines given off by the bacteria. The sudden manner in which many of the acute infections start would lead one to believe that the body, by means of its cells and fluids, can offer resistance up to a certain point by counteracting in some way or other the multiplication and activity of the invading germs.[1] After a longer or shorter interval the balance between the attack and defence is suddenly lost and the symptoms of the infection suddenly appear. As the infection progresses, various substances are elaborated by the resisting organism which possess germicidal and antitoxic properties, and the invading germs are by degrees, or in some diseases where there is a well-marked crisis suddenly, killed off or rendered harmless and their toxines are neutralised. In some cases, however, the reaction of the infected individual may be below the average, or, on the other hand, the infection may be more severe, and the toxines not being neutralised the individual succumbs to toxæmia and dies. It must be remembered that death may also occur from lesions caused by the presence of the bacilli, as for instance death by suffocation in laryngeal diphtheria, or by hæmorrhage or perforation in typhoid fever.

The amount of "resistance" offered to various infections by different individuals may vary considerably; that is to say, there may be an actual *predisposition* on the part of the individual to certain infections. This predisposition may be "natural,"—that is to say, that certain species of animals and certain races of men are particularly prone to develop certain infections,—that is to say, their natural resistance towards these infections is below the normal. On the other hand, predisposition may be artificial or acquired. Many diseases are more easily contracted by persons whose power of resistance has been weakened by starvation, fatigue, loss of blood, damaged digestive power, or exposure. In such cases the vitality of the individual is so depressed that germs which would find no chance of obtaining a lodgment in the tissues in a state of health, easily succeed in infecting him.

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[1] See "Immunity," p. 158.

On the other hand, as opposed to predisposition, there may be *immunity* to an infection. This immunity, also, may be either natural or acquired. Natural immunity is shown in the cases of individuals who, frequently exposed to an infection, fail to contract it. It may be in all probability inherited. The fact that the father and ancestors of a person have all suffered from a given infection may act as a protection against the child contracting that infection badly or contracting it at all. The converse of this is seen in the fatal ravages caused by such a disease as measles, when it is introduced into a community in which it was previously unknown. Epidemics in the Fiji and in the Faroe Islands are instances of how serious a comparatively mild disease may be when it obtains a perfectly new soil. The immunity of an animal to infection may be complete, that is to say, certain species appear incapable of contracting certain infections, or it may be partial, in which case the power of resistance to a certain infection appears increased, and the disease, if caught at all, runs a mild course. This natural immunity has been variously attributed to (α) phagocytosis, (β) to the presence in the blood of certain germicidal bodies called "alexins," which are of a different nature from antitoxins, and which can be separated from the blood and tissues of most animals, and (γ) to a vital reaction of the tissues at present unexplained. This subject is fully discussed in the article "Immunity."

Immunity may be "acquired" by a previous attack of the same infection. That is to say, one attack of an infectious disease usually protects against that disease for life, or at least for a considerable period. This immunity is of course acquired during the attack of the infection, and it is its establishment that leads to a favourable termination. It may also be conferred by vaccination or by the injection of antitoxin prepared from the serum of an immunised animal. Such antitoxin may be not only prophylactic in its nature, but actually curative, and it is on this that treatment by serum is based.

It must be remembered that an infection is not always due to one germ only. There may exist what are called "associated infections," where the illness of the patient is due to the presence and action of two or more species of micro-organisms in combination. Thus clinical diphtheria may be not merely the infection of a mucous membrane by Löffler's bacillus. With that bacillus may be associated various other forms of micro-organisms, such as, notably, the staphylococcus or the streptococcus. And a throat which is infected by these septic organisms is very often the starting-point of a toxic infection more severe than when merely a pure culture of the bacillus is present. In fact the more severe cases of diphtheria are those associated with other micro-organisms. In the same way in inoculation experiments on animals it is found that certain microbes are far more virulent in their specific action if other micro-organisms are injected with them.

Further, two or more pathogenic germs can exist in the human subject concurrently, and independently of each other produce the characteristic lesions and symptoms associated with each. In other words, *coexistence of infectious diseases* is quite possible, and, although it is a possibility which is too often disregarded, is comparatively common. The old view that two infections cannot exist together is quite an error, and any one with any experience of infectious disease has seen scores of cases of concurrence. One of the most frequent is the concurrence of scarlatina and diphtheria. The two diseases may exist together from the same date, or one may follow closely or tardily on the other. Again, a combination of scarlatina and measles in the same patient is by no means rare. And cases have been noted in which scarlatina, diphtheria, measles, and varicella have practically coexisted.

THE PROPHYLAXIS OF INFECTION.—To combat infection the most efficient method is care of the general sanitation in the first place, and of the health of the individual in the second. Remembering the various ways in which infection may be carried to the individual, it is the duty of the community to secure that he should live in a pure atmosphere, have a water and milk supply that is above suspicion, and that the cleanliness of streets and houses should be made an object of rigid care. Should infection break out, the local authority depends on, firstly, *notification*, a system under which every practitioner who diagnoses a case of infectious disease is bound to notify the medical officer of health, which enables that officer to inquire into the cause of the outbreak and take the necessary measures for preventing its spread. Secondly, it depends on *isolation* of the infected person either in a fever hospital or in suitable rooms in the patient's own home. The latter plan is seldom safe unless the house is large and airy, and the whole top flat can be placed at the disposal of the invalid. Thirdly, in certain more dangerous diseases, such as plague or typhus, *quarantine* may be imposed on persons who have been in contact with the sufferer. Fourthly, in some diseases *protective inoculation*, such as antitoxin in diphtheria, or vaccination in small-pox, may be offered to the so-called "contacts." In addition to these steps great attention must be paid to *disinfection*, and the reader is here referred to the article under that head (vol. iii.).

For the benefit of medical officers of schools the following rules will be found useful:—

RULES FOR THE PREVENTION OF INFECTIOUS DISEASE

1. The following diseases are considered infectious:—Whooping-cough, diphtheria, measles, scarlet fever, German measles, small-pox, typhus fever, typhoid fever, chicken-pox, mumps, ringworm.

2. At the beginning of each term, certificates, which will be duly sent to parents and guardians, are required to be shown to the headmaster on the entry or return of pupils, signed by the parents or guardians, not earlier than the day before admission, stating that to the best of their knowledge the pupil has not, *for at least three weeks*, been exposed to any infectious disease, nor entered any house where such disease has existed.

3. If a pupil take an infectious complaint, or has been in any way exposed to infection *during the holidays*, parents or guardians must refer to this list of rules, and if the case come within their scope must communicate with the headmaster.

4. If a pupil take an infectious complaint, or has been in any way exposed to infection *during the term*, notice must at once be sent by the parents or guardians to the headmaster.

5. With regard to Rule 2, no pupil will be allowed to enter or return to school without the certificate signed by *parent* or *guardian*. In the case of a pupil who comes under Rule 3 or 4, he will not be allowed to enter or return to school without a printed form, signed by a *doctor*, to the effect that the said Rules have been complied with.

6. Periods of absence for pupils who have been suffering from any of the above diseases:—

Whooping-Cough.—Six weeks from the commencement of the whooping, provided the characteristic spasmodic cough and the whooping have ceased.

Diphtheria.—In not less than three weeks, when convalescence is completed,—there being no longer any form of sore throat, nor any kind of discharge from the throat, nose, eyes, etc., and no albuminuria.

Measles.—In not less than three weeks from the appearance of the rash, if all desquamation and cough have ceased.

Scarlet Fever.—In not less than six weeks from the appearance of the rash, if desquamation have completely ceased, and if there be no discharge from the nose, ears, glands, or any suppurating sore.

German Measles.—In not less than two weeks from the appearance of the rash.

Small-pox.—One week after the crusts have all separated.

Typhus Fever.—When convalescence is complete, five weeks.

Typhoid Fever.—In not less than six weeks from the commencement of the disease, or of a relapse, if such occur.

Chicken-pox.—One week after the crusts have all separated.

Mumps.—Three weeks from the beginning of the swelling, if all complications have subsided.

Ringworm.—(*a*) *Of Body*—One week after the disappearance of the characteristic eruption. (*b*) *Of Scalp*—One month after the pupil has been certified by a medical man to be apparently free of the disease. The pupil must also be re-examined within two days of return to school, and bring with him a certificate that he is still free of the disease.

In addition to these various periods the patient must in all cases be thoroughly disinfected (see vol. iii. p. 33).

7. In the case of a pupil being exposed to infection, the following quarantine times may be considered safe if *thorough* disinfection be carried out:—

Whooping-Cough	21	days'	quarantine.
Diphtheria	10	,,	,,
Measles	16	,,	,,
Scarlet Fever	10	,,	,,
German Measles	21	,,	,,
Small-pox	18	,,	,,
Typhus Fever	21	,,	,,
Typhoid Fever	21	,,	,,
Chicken-pox	20	,,	,,
Mumps	26	,,	,,

Ringworm:—(*a*) A pupil who has been casually exposed to the infection of ringworm must be kept at home and carefully examined daily for ten days before being sent to school. Should any suspicious spot appear during this time medical advice must be taken before his return. (*b*) No pupil shall attend school from a house in which there is a case of ringworm *of the scalp* within a fortnight of the detection of the disease, and then can only attend if he brings with him a medical certificate that he is free from ringworm himself, and that he has been scrupulously isolated during this time from the case under treatment.

N.B.—As the risk of infection in this variety of ringworm is much less in the case of older pupils (say over 15), special exceptions may be made in their case on communication with the headmaster or headmistress.

8. The fact of a pupil's having had any of the above diseases does not exempt from the above periods of quarantine, except in cases of whooping-cough, German measles, chicken-pox, or mumps, in which cases a pupil may enter or return to school after one day's quarantine.

9. No pupil shall enter or return to school from a house in which there has been any person suffering or convalescent from any of the above infectious diseases, until such house has been thoroughly disinfected to the satisfaction of the medical man in attendance; and it must be borne in mind that the quarantine must date from the time that such disinfection was completed.

10. No article of any kind which has been worn or used by any person suffering from infectious disease, or has been in contact with any such patient or his attendant, shall be brought to school, unless it has been dealt with in accordance with the suggestions for disinfection given (*q.v.*).

Note.—Influenza must be considered an infectious disease, but as yet no definite rules regarding the length of quarantine and of the infective period are formulated. Meanwhile it is recommended that any pupil who has had influenza should be thoroughly convalescent before returning to school; and that pupils from a house in which there is a case of influenza should be isolated from the patient, and should be kept at home if they show the slightest sign of illness.

Inflammation.

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Since the introduction of the term inflammation to indicate the cardinal symptoms of redness (rubor), swelling (tumo), heat (calor), and pain (dolor), which characterise the inflammatory processes when seen upon the skin or other similarly exposed part, its meaning has undergone so many additions and modifications, that it is difficult to find any two pathologists agreeing upon its significance. A definition which would be satisfactory to all is impossible, and so much at variance are their views as to its nature, that unity of opinion is not likely ever to be attained. Some would restrict it to mean only a local disturbance of nutrition accompanied by exudation from the blood-vessels, while others would extend it to include so many other changes that it becomes practically synonymous with local disease. Owing to the differences of opinion being so wide and practically irreconcilable, it is thought by many that the term inflammation should be discarded from pathological nomenclature. This view is probably scientifically correct, but it is not likely to prevail. So much valuable work has been done under the time-honoured title of inflammation, and so rich is it as a storehouse of knowledge, that it will not be easy, even if it were wise, to displace it from its high position in pathological literature. In its earlier and simpler acceptations it includes a series of phenomena of pre-eminent importance in the study of many diseases pretty generally recognised and understood by pathologists. It is important to note at once that it is not a simple vital process, but a combination of different vital processes which may be conveniently studied and demonstrated by an experiment similar to that originally performed by Cohnheim upon the web of the frog's foot. As modified by Coats, it is so easy of performance and its phenomena so readily observed, that it has long been with teachers the favourite method of introducing the subject to their students. The frog should be pithed and curarised. Its foot, with the web spread out, is fixed upon an ordinary microscopic glass slide, so that it may be examined under the microscope. A small portion of the surface of the web is snipped off with scissors, just deep enough to remove the epithelial layers without drawing blood, and the subsequent inflammatory processes carefully observed. They are as follows: 1. Active Hyperæmia and Acceleration.—The arteries in the immediate neighbourhood of the injured part dilate, then the veins, and the rate of blood-flow therein is obviously accelerated. These changes last a considerable time, and are followed by others generally in about an hour. Many capillaries formerly invisible begin to appear and become distended with blood. 2. Hyperæmia and Retardation.—After a time the rate of blood-flow becomes slower, and continues to do so until it is slower than normal. The vessels, particularly the capillaries and veins, instead of showing the usual well-marked distinction between axial stream of corpuscles and peripheral stream of plasma free from cells, show a mixing of these streams. The axial one broadens out and gradually encroaches upon the peripheral one. Then leucocytes fall out into this peripheral stream and move slowly along, every now and then becoming adherent for a moment to the vessel wall only to move slowly onwards again at intervals. They may even regain the axial stream. Blood-plates often also appear in this marginal zone. The blood-flow gradually becomes still slower, and red blood corpuscles pass out into this same zone, and soon all distinction between axial and peripheral streams is lost. The lumen is filled with a mass of red and white corpuscles whose shape and outlines are now distinctly seen. The latter gain the wall of the vessel in increasing numbers, and adhere firmly to it, generally becoming pear-shaped owing to the pressure of the blood-flow. The slowing continues until the blood is moved onwards merely by a succession of jerks, or even oscillates slowly backwards and forwards, ultimately coming to a complete standstill, a condition of stasis. 3. Exudation of Fluid.—A considerable oozing of clear fluid now comes from the wound. It began earlier, but has now become marked. It comes from the distended vessels, particularly the capillaries and veins, and is generally spoken of as an outpouring of lymph. 4. Escape of Blood Corpuscles.—The leucocytes adherent to the vessel walls gradually make their way through until they lie altogether outside the vessels. It takes a leucocyte a very considerable time to emigrate from a vessel, probably because it has to squeeze itself through an aperture greatly smaller than itself. When half through it may be seen as a rounded swelling both outside and inside the vessel, connected by a very narrow thread. Emigration occurs most abundantly from the small veins, but it also occurs to some extent from the capillaries. Although the wall of the latter is so much thinner, yet the lumen is so narrow that the same margination and accumulation of leucocytes cannot take place. The process generally continues steadily for several hours, until a great concourse of leucocytes showing active amœboid movement can be seen lining the outside of the veins and in lesser numbers the capillaries. Little groups of red blood cells may also be seen in places among the leucocytes. The passage of the red cells through the vessel walls is known as diapedesis, a term which has been extended by some authorities to include also the emigration of the leucocytes. Many of the latter move from the neighbourhood of the blood-vessels towards the injured surface, and they may escape in such numbers in the exudate that it becomes opaque instead of clear. Others break down, and fibrin appears in the exudate, so that the gap becomes filled with a colourless clot which overlaps the edges. 5. Reaction of the Fixed Tissue Cells.—The epithelial cells at the margin now begin to proliferate and grow over the wound towards one another. Circulation begins afresh in the affected vessels, slowly at first, but steadily and gradually. The vessels contract, the leucocytes disappear, and soon (generally in about three days) the circulation appears to be perfectly restored to its normal state. The proliferation of the epithelium has meanwhile continued and is completed about the same time, so that now all traces of the injury have disappeared. These later stages cannot be seen in the pithed frog, and hence fresh animals must be used; and further, the greatest care must be taken to protect the wound from all contamination. The mesentery or tongue of the frog may be chosen for experimentation instead of the web. The mesentery of the cat, dog, etc. or the ear of the white rabbit, or wing membrane of the bat, are also suitable structures, as they all permit the direct observation of the circulation under the microscope. Exactly similar changes are seen in naturally occurring inflammations of the skin and serous surfaces, such as the peritoneum, pleura, meninges, and joint surfaces, etc.

The Causes of Inflammation.—The exciting cause of the inflammation in the foregoing experiment is a mechanical one. Chemical, thermic, and electrical influences act in a similar manner. Thus the mere exposure and fixation to the microscopic slide of the mesentery of a warm-blooded animal is sufficient to set up a severe inflammation. The rapid cooling and evaporation must be held in check otherwise necrosis may set in. The mechanical, thermic, chemical, and electrical agents are classed as external noxæ, and to them must be added the most important of all, viz. infections in which bacteria are the causal factors. A combination of noxæ may occur. Thus bacteria may gain a footing in a tissue already inflamed through the action of another agent. The mechanical, electrical, and thermic noxæ must always act externally, causing inflammation at their site of impact, whereas bacteria may enter the body without setting up any change at their point of access, and being transported by the blood or lymph may cause inflammations at other and distant parts of the body. Such inflammations are metastatic, and the noxæ may be called internal. Bacteria act through the agency of their products (chemical substances) technically known as toxines. They may be formed by bacteria within the body, or may be preformed and introduced as such into the body, *e.g.* in poisonous tinned meats. It is to be remembered also that other injurious chemical substances may be produced within the body entirely independently of bacteria, which may act in the same way. This may occur in functional disturbances of several of the internal organs, such as the liver, kidney, intestines, etc. These abnormal products of metastasis may be absorbed by the lymph or blood, and set up metastatic inflammations exactly as the toxines of bacteria do. Any noxa, external or internal, in order to cause inflammation must be of a certain degree of intensity. If too severe it will lead to the death of the tissue, and then vital processes are no longer possible.

THE PHENOMENA OF INFLAMMATION.—The ordinary processes of inflammation as seen in the foregoing experiment and similar lesions may be arranged as follows: 1. Active hyperæmia and acceleration; 2. Hyperæmia and retardation; 3. Escape of fluid; 4. Escape of blood corpuscles; 5. Changes in the fixed tissue cells. The cardinal symptoms of redness, heat, swelling, and pain are appreciable by the unaided senses, and are easy of explanation. The redness is due to the greater amount of blood in the part, the heat to the rapid transference of the hotter internal blood to the surface, and an increased flow of blood through the part. It is only seen in external, not in internal inflammations. The swelling is due to the distension of the blood-vessels and the exudation; the pain (to which functional disturbance may be added) to the increased pressure upon the nerve-endings, to the impeded circulation and nutrition, and partly also directly to the exciting cause or noxa. A closer examination aided by the microscope reveals the other changes just mentioned which are invisible to the naked eye, and include these coarser microscopic changes. They may be studied in detail.

I. *Active Hyperæmia and Acceleration.*—The dilatation of the arteries is brought about partly reflexly either by a paresis of the vaso-constrictor nerves or a stimulation of the vaso-dilators, and partly by a direct paralysing action on the arterial and capillary walls and the connective tissues surrounding them. It may pass off without the appearance of other inflammatory phenomena, and the latter may appear without its preceding them. Hence it is not an indispensable process, but a more or less fortuitous forerunner of inflammation.

II. *Hyperæmia and Retardation.*—The dilatation of the veins and capillaries follows upon that of the arteries, and increases. Retardation of the blood-current follows upon the acceleration. The cause is the alteration of the vessel walls and surrounding tissues, produced by the action of the noxæ, especially those tissues in the neighbourhood of the capillaries. These changes will be presently described under the reaction of the fixed tissues. They lead to increased friction between the blood and the vascular walls, so that the red and white corpuscles escape into the peripheral stream. The blood-plates, which may also fall out, are held by some to be normal constituents of the blood, and by others to come from degeneration and breaking down of the red cells. These processes are practically always observed in all inflammations of vascular tissues, and hence may be regarded as essential phenomena of such inflammations.

III. *Escape of Fluid.*—There is a liquid transudation from the blood-vessels similar to the normal lymph transudation, but the fluid differs in amount and composition from the lymph. It is more abundant, more albuminous, and contains a greater amount of solids. A deposit of fibrin sometimes occurs. The cause of these differences is the alteration brought about in the vascular walls through the action of the noxa. The normal selective power of the vascular walls is not lost, for the altered walls still act as a secreting and not as a mere filtering apparatus. The alterations in the blood-pressure may greatly assist the transudation process, but the alterations in the vascular walls are the primary cause. The exudate infiltrates the fissures of the surrounding tissues and may cause considerable swelling. When it finds its way on to a free surface, such as a mucous or serous surface, it may escape or collect in a serous cavity. Part of it may be absorbed by the cells and other tissues, which may consequently show coarse granulation, mucoid, and other degenerations. They may even undergo complete disintegration, and their elementary constituents may dissolve in the exudate. The chemical composition of the fundamental substances in the exudate may also undergo change through their contact with the tissues of the part. The exudate always contains cells, and if it be very rich in leucocytes it is called pus. It may also contain large numbers of red corpuscles when it is spoken of as hæmorrhagic. The exudate may be taken as a measure of the severity of the inflammation, for usually the greater the severity the greater the exudate, and the more prolonged will the inflammation be. Its amount and character, therefore, depends largely upon the nature and degree of intensity of the noxa, but also partly upon the region affected and the condition of the tissues. It is an essential phenomenon of inflammation in vascular tissues, and is never really absent therefrom. While often a useful process, it is by no means always so. When serous it may act a beneficial part by flushing out the injured area and diluting the noxa. This will be advantageous, especially upon external surfaces and mucous membranes, as a large quantity of serous fluid may wash away the poisons or at least weaken their effects. When fibrinous it may circumscribe the inflammation by preventing the spread of the irritant. This is well seen in the fibrinous adhesions which so frequently localise an appendicitis or a pelvic peritonitis, and even in more diffuse forms, such as pericarditis, it may do good by closing the stomata and interfering with toxine absorption by the lymphatics and blood-vessels. The exudate may also contain bactericidal and digestive substances, leading to the production of peptones and the death of bacteria. It may lead to increased nutrition of the affected region, which may be useful in the later, though rarely in the earlier stages of inflammation. On the other hand, the exudate is positively hurtful when it collects in the body cavities and ventricles of the brain, and when it accumulates in the lung alveoli, or in the pharynx and air passages. Further, instead of proving injurious to bacteria, it may be actually helpful by providing them with a good nidus for their further growth and development.

IV. *The Escape of Blood Corpuscles.*—1. The Leucocytes.—Their escape from the blood-vessels is an active vital process. The first step is their margination along the wall of the blood-vessels, especially the small veins. It is brought about by action of the noxa upon the vessel walls, as it never occurs in a healthy vessel. It is a deteriorative or degenerative action, generally demonstrable histologically. The nucleus of the endothelial and other cells becomes rounded, the protoplasm granular or vacuolated, or mucoid, colloid, or other degeneration appears. Even in the exceptional cases in which there is no detectable histological change there is always a disturbance of the perfect endothelial pavementation of the lumen. The cement substance between the endothelial cells is deranged and loosened. It becomes irregularly distributed along the lumen and widened in places, as can be shown by treating it with the silver nitrate process. The amount and degree of this change will depend mainly upon the nature and intensity of the noxa, but it is rarely so great as to cause the appearance of actual visible pores. They exist potentially, and the leucocytes, after margination, become adherent to the endothelial cells, and commence to push a part of their protoplasm through between them at intervals here and there along the vessel wall, and the entire cell body gradually follows. In the capillaries, where the process can be most easily studied on account of the thinness of their walls, the leucocytes are always seen to pass out between and never through the bodies of the endothelial cells. It is chiefly the polymorpho-nuclear leucocytes, comprising roughly 70 per cent of the total number of the corpuscles present in the blood, which emigrate. They pass out of the vessels through their amœboid movement, and may in the same way transport themselves to considerable distances. The activity of this amœboid movement appears to be largely dependent upon the nature and intensity of the noxa. The more the blood-vessel wall is deranged the more readily can it occur. They do not show it while still within the vessels and previous to adhesion to their walls. It has been suggested that substances are formed in the tissues in inflammation which attract the leucocytes, and the term positive chemotaxis has been introduced to designate this attraction. Many substances can be experimentally shown to exert it, *e.g.* thin solutions of glue, of the gluten of wheat, and liquids containing the products of bacterial action. Substances having this positive chemotaxic action may be produced from various sources in inflammation, such as the products of bacteria and the products of degeneration of the fixed tissues. The margination and adhesion of the leucocytes are apparently entirely due to the degenerative changes in the vessel walls, uninfluenced by chemotaxis. Nor can the latter be regarded as the cause of either the beginning or the continuance of the amœboid movement, since leucocyte emigration may take place when it is certain that no such action is being exerted. It cannot be more, then, than an active and important stimulant thereof. Certain substances may arrest or prevent the emigration of leucocytes, *e.g.* solutions of quinine, iodoform, salicylic acid, and a 1½ per cent solution of common salt. This action is capable of different explanations. It may be attributed to a negative instead of a positive chemotaxis, causing an inhibition of amœboid movement, or to a contraction of the vessel walls which interferes with their permeability and with the adhesion of leucocytes. Such substances are not, therefore, suitable washes or applications for wounds where free leucocyte emigration is desired. Leucocyte emigration occurs most abundantly in inflammations set up by the pyogenic bacteria, viz. the staphylococci and streptococci, which cause what are known as suppurative inflammations or suppurations. They have been most carefully studied in their experimental production in the skin and cornea, and in their natural occurrence in various organs and tissues. The leucocytes emigrate extensively from the blood-vessels, and collect chiefly around and among the cocci. Many of them have their protoplasm crowded with cocci which they have ingested. This engulfing of the cocci by the leucocytes, which are consequently called phagocytes, is an active process, and is held by Metschnikoff and his followers to be of vital importance in controlling, curbing, and counteracting the pyogenic inflammatory processes, by gradually bringing about the destruction of the causal bacteria. This is the doctrine of phagocytosis. It may be carefully studied in the experimental production of abscesses. There is first an accumulation of lymphocytes in the affected area, and then emigration of polymorpho-nuclear leucocytes from the neighbouring blood-vessels occurs. They collect around the cocci, which are meanwhile multiplying rapidly, and being largely ingested by many of the leucocytes. The liquid exudate from the blood-vessels collects in the tissue spaces, and may help the excretions of the cocci (the main agents) to digest and liquefy the formed tissues of the part. Upon a free surface the liquefied tissues escape and leave an ulcer. In a deep-seated part they collect as a liquid mass containing cocci, degenerated leucocytes, and tissue debris, and form an abscess. The liquid contents are called pus. Bacteria are generally present in acutely formed abscesses, but often absent, having disappeared in chronic ones. They are always seen in greatest numbers at the margins of the abscess, and it is here also that phagocytosis is most active; and further, so long as the suppurative process is extending, the cocci are observed in the tissue spaces outside and beyond the leucocytes, whereas the reverse is the case when it has ceased to spread. It is the polymorpho-nuclear and the large hyaline uninuclear leucocytes which act as phagocytes. It is generally held that the coarsely granular eosinophile leucocytes, which are present in very small numbers in the normal blood, but plentifully in the tissue spaces, never act as phagocytes. The writer has, however, seen them crammed with gonococci in gonorrhœal discharge. The bacteria ingested by these various phagocytes can in many cases be shown to be still active and virulent, and capable if liberated of exerting their specific action, while in other cases they are degenerated and inert, and dying or dead. Relying upon this and similar arguments, and particularly upon the reactions to irritation exhibited by leucocytes in the lower animal structures, such as the tadpole's tail and tissues of the invertebrata generally, the opinion that phagocytosis is always a beneficial process has received much support from pathologists especially in recent times. It has been likened to a struggle between the leucocytes and bacteria, in which both strive for the mastery. If the former should prove victorious the disease will be arrested and cured, and *vice versa*, the disease will continue and extend. This doctrine can no longer be upheld. It is undoubtedly true that phagocytosis may be a beneficial process. This is seen in many of the acute suppurative inflammations caused by the pyogenic cocci, and in other instances where the leucocytes ingest and destroy active and vigorous bacteria, as in infectious catarrhs of mucous membranes, where the leucocytes, by migrating to the surface, remove the bacteria from the affected part. On the other hand, it has been proved that the power of killing bacteria does not rest with the leucocytes alone, but is possessed in a much higher degree by the blood serum, lymph, and tissue fluids generally, and also by the fixed tissue cells which may act as phagocytes. Further, the leucocytes ingest solid particles of all kinds seemingly as readily as germs. They can be made to ingest nutritive particles, fat globules, pigment granules, red blood corpuscles, etc., by mere physical contact therewith, or after stimulation with various chemical solutions. Therefore the suggestion that phagocytosis is fundamentally of a nutritive nature seems as reasonable as that it is protective. In many cases, indeed, it can be shown to have a hurtful and not a beneficial action, as when leucocytes containing virulent bacteria migrate to healthy parts and there form a new focus of disease. The mere imprisoning of the bacteria within the protoplasm of the leucocyte does not render them harmless, as they may not only preserve their virulence intact, but increase it, since the leucocyte protoplasm may form a suitable and favourable bacterial nidus. Phagocytosis, while often playing an important part, cannot be held to be an essential phenomenon of inflammation, as it may be entirely absent in typical instances thereof. The leucocyte emigration is a wider phenomenon. It leads to an accumulation of leucocytes in the inflamed area which may be conveniently spoken of as a leucocytosis. The amount of leucocytosis varies within wide limits, being dependent upon several factors, the chief of which is the nature and intensity of the noxa and the anatomical structure of the affected part. While emigration from the blood-vessels is the chief source of the leucocytes, some, never many, come in certain cases from the wandering cells of the tissues, and others from an actual division of leucocytes *in situ*. They are rarely if ever produced by the transformation of fixed tissue cells, although this has been said to occur by more than one observer through the degeneration of muscle cells. According to the more restricted conception of inflammation, the presence of blood-vessels, either in an area or near it, is necessary for the possibility of the production of inflammation, and inasmuch as leucocyte emigration is the chief source of the leucocytosis which constantly occurs in its establishment, it must be regarded as being one of the essential phenomenon of such inflammatory processes. According to the wider acceptation of inflammation, which holds that blood-vessels are not at all necessary, the leucocytosis becomes still more important. The cornea is the structure which has been most widely chosen for experimentation in this relationship, for it is anatomically a non-vascular tissue. Its marginal parts (sclerotic) are so plentifully supplied with blood-vessels, that it is hardly possible to set up irritation in its central parts without transmission to these peripheral vessels. Therefore it is practically a vascular tissue. In the invertebrate kingdom, on the other hand, the tissues may all be regarded as non-vascular, as there is no closed system of blood-vessels. They have, accordingly, been selected by many workers as affording a wide field for experimentation and investigation. Without entering into particulars, it may be stated generally that a more or less marked leucocytosis always appears in the irritated area. In many of the lower forms it seems to be the dominant factor, many of the leucocytes afterwards assisting in the process of repair. In the higher forms the leucocytosis is still very prominent, but the process of repair seems to be relegated entirely to the fixed tissue cells. Leucocytosis, therefore, appears to be an invariable and essential phenomenon of inflammation, whether the wide or restricted conception of the term be adopted. What purpose does it serve? We have already seen that many of the leucocytes act as phagocytes, and that it is at least probable that phagocytosis has a nutritive rather than a protective value. It is possible that this function is possessed by all the leucocytes, though other functions are also possible. Many of the leucocytes degenerate and break down, producing substances which dissolve in the tissue fluids and prove hurtful to bacterial life and activity. They may, in other words, through degeneration or otherwise be largely instrumental in producing substances which confer bactericidal properties upon the body fluids. Others may produce fibrin ferment and lead to the production of fibrin threads or masses within the inflamed area. Further, when recovery is taking place, solid masses of fibrin must be dissolved previous to their absorption, and this must be done largely through the agency of cells which may be leucocytes.

There is still another possible destiny of leucocytes which must be carefully considered, viz., the transformation of leucocytes into fixed tissue cells. There is no evidence whatever that this occurs in the case of the polymorpho-nuclear leucocytes, but with the mono-nuclear forms it is not so clear. These may leave the blood-vessels in considerable numbers, and collect in the affected area in the more chronic forms of inflammation, in which we subsequently get a marked fibrous tissue formation or fibrous hyperplasia. It has not yet been shown, however, that the mononuclear leucocytes contribute in any way to this formation. Histological methods have been so improved of late that it is possible to differentiate clearly the various cells of a tissue in the many changes which they may undergo, and so far no absolute proof of the conversion of mono-nuclear leucocytes into connective-tissue corpuscles has been brought forward. Metschnikoff and others have claimed that this does occur in the lower tissues of animal life, as in the tadpole's tail, and although it has never been shown to exist in the higher forms, yet it must be held to be possible, as it has never been definitely excluded. 2. Escape of the Red Blood Corpuscles.—This is a purely passive process dependent primarily upon the change in the vessel walls, and hence is usually well marked in severe inflammations. The nature of the irritant has also considerable influence, some irritants causing it much more readily than others. The greater density of the blood owing to the escape of the exudate, the adhesiveness of the lining membrane of the vessel walls, and the blood pressure, may all assist the process. The escape of the red blood cells from the blood-vessels is known as diapedesis. It subserves no special function. 3. Escape of the Blood Plates.—This takes place about the same time as the escape of the other corpuscular elements. It is somewhat difficult to follow, and comparatively few observations have been recorded, but so far as we know it has no significance in inflammation.

V. *The Rôle of the fixed Tissue Cells.*—The fixed tissue cells play an important part in all inflammations. The changes which they undergo depend mainly upon the nature and degree of intensity of the irritant. They may be divided into two stages, a degenerative or retrogressive stage and a regenerative or progressive one. 1. The Degenerative Stage.—This varies within wide limits, sometimes showing swelling and granulation of the protoplasm, and sometimes fatty, mucoid, vitreous, or other obvious degeneration. At other times the changes are so slight that they cannot be detected histologically, but they are always sufficient to cause disturbance of function. The matrix also undergoes degenerative changes. It loses its elasticity, and fails to exert the normal pressure upon the walls of the blood-vessels. Its resisting power is diminished, and it may show the same obvious degenerations as the cells, or even be dissociated and liquefied. Hyaline degeneration is common in chronic forms of inflammation. The direct action of the noxa is the main cause of these changes, but the absorption of exudate by the cells and tissues may greatly assist it. The exudate is often rich in mucin, which causes swelling, softening, and mucoid degeneration of the tissues. The fixed tissue cells also become detached. In the case of the connective-tissue cells they separate from the fibrous bundles, and in the case of gland tubes from their attachment to the basement membrane or wall of the tubes. The liberated cells may break up and disappear, or they may show signs of division and ultimately lead to multiplication. 2. The Regenerative Stage.—The newly-formed cells may produce new tissue after their own kind, and thus bring about repair of lost tissue. This can be well studied in the cornea. If its central part be injured, *e.g.* by a solution of zinc chloride, a loss of tissue arises at the injured point. The blood-vessels at the margin of the cornea dilate, etc., and give rise to an exudate. Leucocyte emigration and the other typical signs of inflammation are duly set up. After a time changes are seen in the corneal corpuscles surrounding the injured area. They are seen to swell and proliferate, producing new cells. Some of them attain a considerable size, producing large cell masses each with many nuclei. The exudate and leucocytosis subside gradually, the peripheral blood-vessels return to their normal condition, and the newly-formed corneal corpuscles proceed to form new fibres, and in a short time all signs of the corneal injury have disappeared. This rapid and perfect healing is usually well exemplified in corneal ulcers. Much the same changes occur in vascular tissues, but the blood-vessels generally take a more active part, especially in considerable inflammations. The lining endothelial cells are about the first structures to be affected by the noxa, and it is only after they have been effected that escape of exudate and leucocyte emigration become possible. Later they may, like the connective-tissue cells outside, become detached and proliferated. The exact time at which the degenerative stage ends and the regenerative begins does not permit of delimitation. A certain time must elapse before regeneration can commence (five to twenty-four hours or more), and it is rarely of any extent until after the third day, the time required being largely dependent upon the character and intensity of the irritant. In the same way it may be indeterminate in its ending, and result in various forms of hyperplasia. This is especially seen in chronic inflammations. The degenerative stage may be very slightly marked, while the regenerative is very pronounced. The interstitial fibrous tissue which is so abundantly formed in cirrhosis of the liver and kidney is a good instance in point. The new formation of fibrous tissue is here looked upon as inflammatory, since it is due to the continued action of some irritant. On the other hand, somewhat similar changes may arise entirely independently of inflammation. In granular contracted kidney there is primarily a degeneration and atrophy of the renal epithelium, ending in its destruction over certain areas, and keeping step therewith there is a compensatory overgrowth of the fibrous interstitial tissue. Inflammation has nothing to do with these changes, although they give a renal cirrhosis practically indistinguishable in its later stages from that produced by inflammation. In the same way the regenerative processes of inflammation must be carefully distinguished from those of repair. They are not synonymous, though it is by no means easy to distinguish between them when they are present together, or when the one follows at once upon the other. This does not necessitate the exclusion of the healing processes from inflammation. When the blood no longer carries injurious substances, or when the influence of the irritant is at an end, normal blood will circulate through the affected vessels, conveying healthy nourishment to their walls and the tissues around, and thus gradually restore them to their healthy state (the *vis medicatrix naturæ*). The exudate is absorbed, the leucocytosis disappears, and in slight cases the tissues regain their normal structure and functions, but in more severe cases there is an actual loss of tissue which must be restored. There is a general tendency for lost tissue to be replaced by tissue after its own kind. When the new tissue is exactly like that which was lost the healing is perfect, but this rarely occurs in the highly specialised tissues. Bone and ganglion cells do not proliferate at all, and cartilage cells only very slightly. Striped muscle cells have considerable powers of proliferation, except in the case of the heart where there is none. Non-striped muscle cells proliferate comparatively freely, and so do neuroglia cells and glandular epithelial cells, while covering epithelial cells, ordinary connective-tissue cells, and endothelial cells, have great and rapid proliferative powers. Many losses of tissue are consequently replaced by an inferior form of tissue. In the heart, for example, a loss of substance is replaced by fibrous tissue. This occurs also largely throughout the internal organs. Connective tissues in the main replace lost structures in the central nervous system (through proliferation of the cells of the vascular sheaths and neuroglia cells), liver, and kidney. Gland cells may be reproduced in slight losses in glandular organs, but they are usually of a low functional value. In more severe losses the gap is made good by fibrous tissue. The remaining healthy gland tissue has great power of compensatory hypertrophy. Nerve trunks, on the other hand, are largely regenerated. If a part of a peripheral nerve be destroyed, fibrous tissue first proliferates and fills the gap, and then the axis cylinders of the central end send out prolongations which perforate the fibrous tissues, and seeking out the distal end of the nerve in time re-establish connection with the peripheral nerve end organs. They acquire new medullary sheaths. In striped muscle fibrous tissue first fills the gap, and later new muscle fibres are formed which penetrate the fibrous scar more or less extensively, but rarely succeed in completely restoring the muscular continuity. Unstriped muscle fibres, on the contrary, are rarely reformed to any extent, though their power of proliferation is considerable. New bone is often extensively produced, but always from the periosteum, endosteum, or perichondrium. Covering epithelium, whether squamous, mucous, or serous, is generally widely restored, and always from proliferation of the pre-existing epithelium, never by any other tissue. The subepithelial fibrous tissues are also extensively and similarly restored by like tissues. The blood-vessels enter into the process of regeneration wherever there has been loss of tissue. New blood-vessels are formed and the new tissue appears around them. This may be easily followed in the healing of an ulcer. Its floor soon becomes covered with little red points which are known as granulations. Each consists of a vascular loop surrounded by cells of various types which may be arranged in three groups: (1) Small round cells with polylobular, fragmented, or single deeply staining nucleus, and with or without oxyphil granules. (2) Large uninucleated cells with a clear protoplasm. (3) Cells of varying size (mostly larger) and shape, with a bright oval nucleus and abundant protoplasm. Their relative proportions vary greatly. The last tends to be deeper than the first two, which are probably both leucocytes. The regeneration of the fibrous tissue is apparently performed entirely by the third type, which is indubitably a connective-tissue cell. It assumes many different shapes, such as spindle, star-shaped, irregular, etc. It is often called an "epitheloid cell." It is seen to secrete fibres either at its side or end. Large cells with many nuclei (giant cells) sometimes appear in granulation tissue. They are epitheloid cells which have become altered by some special character possessed by the noxa, *e.g.* the tubercle bacillus, which can inhibit the division of the protoplasm, but not that of the nucleus. Similar cells are often seen around foreign bodies such as silk ligatures. The new blood-vessels of granulation tissue always arise as buds from the pre-existing capillaries of the inflamed area. They are at first solid (usually) outgrowths which come to bend round and join other similar outgrowths or pre-existing capillaries. They become vacuolated and hollow, and nuclei appear in their walls, which later become segmented into endothelial plates like normal capillaries. The blood soon makes its way through these new vessels from the mother capillaries. The gap of the ulcer will thus in time become filled up by this newly-formed vascular fibrous tissue, and its surface at the same time gradually covered by epithelium. The time which this takes will vary greatly according to circumstances. The most important of all the disturbing factors is the presence or continuance of infection. The amount of inflammatory exudate may be taken as a good practical guide to the probable duration of the inflammation, especially of its later formative phases. The exudate, wherever it is not absorbed by the lymph or blood, must be replaced by granulation tissue, a much slower process. Extensive exudates are followed by prolonged granulation tissue formation, which may lead to extensive fibrous thickenings and adhesions. These conditions hold good for deep (*e.g.* in muscles, bones, brain) as well as for superficial inflammations. Considerable transformation may afterwards take place in the newly-formed tissues. The capillaries may largely disappear and the fibrous tissues increase in number and density. Elastic fibres may appear and the connective-tissue cells greatly diminish in numbers. The ultimate cicatricial tissue is thus often denser, less elastic, less vascular, and more vulnerable than the original tissue which it has replaced. This ultimate development is often slow, and may not be reached for months.

The fixed tissue cells of an inflamed area usually exhibit other changes not referred to above. They may be seen at almost any stage of inflammation to have engulfed leucocytes, bacteria, pigment particles, remains of red blood cells, necrotic tissues, etc. They may do so while still *in situ* or after becoming free. They may show a considerable range of movement. It is probable that they possess and exert a certain degree of selection, for they may be brought in contact with bacteria without at first ingesting them, though they may do so later. This has been explained by the suggestion that the bacteria first exert a negative and later a positive chemotaxis, particularly when inert or degenerated. The phenomenon of phagocytosis is thus exhibited both by leucocytes (microphages) and by fixed tissue cells (macrophages), and if it be of a nutritive value in the former, it is probably so also in the latter.

It is obvious that all the changes which the fixed tissue cells undergo in inflammation cannot subserve a useful purpose, though some of them do. The earlier degenerative changes cannot be beneficial, but many of the later regenerative processes may be. The latter are, however, not always present, or they may be so slight as to have no practical influence. Again, they may be excessive and altogether beyond the requirements of the tissue, and in such cases they may do obvious harm. It is not the necessities of the tissues which call them forth, but the nature and the intensity of the noxa reacting through the vital qualities of the tissues themselves. They are also influenced by the nature and conditions of the structures affected.

NOMENCLATURE USED IN INFLAMMATION.—(i.) *Terms signifying Inflammation.*—It is customary to refer to inflammation of an organ under a term made up of the Greek (usually) name for the organ with the termination itis appended. Hence we say peritonitis, pleuritis, endocarditis, pericarditis, meningitis, bronchitis, etc. The prefixes peri and para attached to these terms indicate that the serous covering or tissues around the organ are inflamed, as perihepatitis, perisplenitis, parametritis, etc.

(ii.) *Duration of Inflammation.*—The course of an inflammation is sometimes short, sometimes prolonged, hence we call it acute, subacute, or chronic.

(iii.) *Kinds of Inflammation.*—The factors which modify inflammation are so many and their interaction so complex, that a satisfactory classification cannot be given. Various groupings are, however, in use, such as those depending on the site, the quantity and quality of the exudate, and the reaction of the tissues themselves.

1. *The Site.*—Those affecting the surface of an organ or tissue, *e.g.* the skin and mucous membranes, may be distinguished as superficial, while those affecting deep tissues, *e.g.* the interior of the brain, bones, etc., are called deep seated. Inflamed tissues near a free surface, *e.g.* skin or mucous membrane, generally show the degenerative changes chiefly in the superficial cell layers, and also an abundant serous exudation (relatively more than into an abscess) which comes to the surface and escapes. It is mixed with cells (epithelial and leucocytes). In glandular organs the inflammation may affect the secreting structures, when it is called parenchymatous, or the supporting connective tissues, when it is called interstitial. Inflammations limited entirely to one or other of these structures are uncommon, but combinations occur frequently. Inflammations of muscular tissue and of the nervous system are similarly distinguished as parenchymatous and interstitial.

2. *The Qualities of the Exudate.*—(*a*) The inflammation is called serous when the exudate is a clear or slightly morbid fluid. It is most often seen in comparatively slight inflammations. (*b*) It is called purulent when the exudate is turbid or whitish by the plentiful admixture of leucocytes. When it occurs naturally in the human subject it may always

be looked upon as caused by bacteria, though artificially, other agents such as turpentine, petroleum, and croton oil may produce it. Suppurations caused by chemicals are not so grave as those caused by bacteria, as they heal more rapidly and do not produce blood poisonings or metastases. (*c*) It is called fibrinous when the exudate undergoes a deposition of fibrin, either as a network, or homogenous, or flaky, or irregular, or granular masses, or as stratified layers. In the lungs it often forms a network, filling the alveoli, and is often spoken of as a croupous inflammation, a term which was originally applied only to the fibrinous deposit, or false, or croupous membrane, which forms upon the pharynx and larynx in certain severe inflammations, particularly in diphtheria. The term croupous was first of all used to describe the peculiar crowing noise of croup, and thence it came to be used to describe the false membrane which caused it. Its extension to other fibrinous inflammations, as in the lung, has nothing to support it, and should be discontinued. In deep-seated inflammations threads of fibrin may be occasionally seen, but its formation is always slight. Large deposits of fibrin are characteristic of severe inflammations of serous surfaces, such as the pleura or pericardium. (*d*) It is called hæmorrhagic when the exudate is mixed with blood, either from a plentiful diapedesis or from an actual rupture of blood-vessels. It sometimes indicates a grave form of inflammation, while at other times it points to some peculiarity of the irritant or of the tissues involved.

3. *The Reaction of the Tissues.*—The degenerative, liquefactive, and necrotic, etc. changes which the tissues show, and which are so largely dependent upon the nature and degree of intensity of the noxa, may also be used to describe special forms of inflammation.

This article does not attempt to give more than an explanation of what is meant when the term inflammation is used, or more than a short account of the various processes involved in its establishment, so as to furnish the busy student and practitioner with a workable and practical view of the subject, unburdened with a discussion of the many problems of a more strictly scientific interest with which its every aspect fairly bristles.

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Definition

Influenza is an acute, specific, infective febrile affection, characterised by its remarkably sudden onset, after an incubation of about two or three days, the many symptomatic phases it presents, the singular predisposition to other secondary infections which it induces, its disastrous effect upon the heart, and the protracted convalescence which follows in its wake. In itself by no means a mortal disease, influenza exercises a far-reaching and malignant effect upon the death-rate, which surges far above the average during the few days or weeks of its epidemic or pandemic prevalence. Catarrhal symptoms, although often present, are not an essential feature of the disease. The victims of influenza are adults, who perish from pneumonia or bronchitis; and the aged, who sink from heart exhaustion. Children, while prone to the disease, enjoy a comparative immunity from its complications and dangers; in them a sharp febrile movement, with or without an evanescent rash of an erythematous nature, constitutes the main feature of the attack. Unlike the poison of typhus, the virus of influenza is not rendered inert by oxidation from contact with atmospheric air, but rather the contrary.

BACTERIOLOGY

The specific micro-organism of influenza was identified by Pfeiffer of Berlin, and independently by Canon of the same city in 1892. It is a very small aerobic, non-motile bacillus, which does not grow at temperatures below 28° C. (82·4° F.) Spore formation has not been observed. Its diameter is $0{\cdot}2\mu$ to $0{\cdot}3\mu$, and it is only about $0{\cdot}5\mu$ in length. It occurs singly or united in chains of two, three, or four elements. It is quickly destroyed by desiccation. The thermal death-point is 60° C. (140° F.) with five minutes' exposure. This bacillus was discovered by Pfeiffer in the gray mucus of the bronchial mucous membrane and in the purulent bronchial secretion; by Canon in the blood of patients suffering from epidemic influenza in 1892. Canon's observations as to the presence of the influenza bacillus in the blood lack confirmation. The deep staining of the ends of the bacillus with dilute carbol-fuchsin (Ziehl) solution or methylene blue (Löffler) solution, with heat, causes it to resemble a diplococcus (Sternberg). Pfeiffer's bacillus is present not only in the nasal and bronchial secretions, but also in the substance of the lung, where it may set up pneumonia in both adults and children. It may be localised in the spleen, pericardium, endocardium, middle ear, central nervous system, and pleura. In severe cases the bacillus is also found in the peri-bronchial and sub-pleural lymphatics (Goodhart).

HISTORICAL

It would be foreign to the object of this article to enter at any length into the history or bibliography of influenza. When preparing his classical memoir on the Mortality of Ireland, published in the Census of 1841, the late Sir William Wilde, M.D., learned many curious facts connected with this disease and its epidemic outbreaks in early times in Ireland. It is described in a medical manuscript of the fifteenth century under the names of *Fuachd* (*i.e.* cold or chillness) and *Slaodan* (*i.e.* a cough or cold), and is mentioned in the *Annals of the Four Masters* as epidemic in Ireland in the fourteenth century. A disease, the symptoms of which answer to those of influenza, is also alluded to in certain early Gaelic manuscripts under the term *Creathan* (*i.e.* a shaking or trembling).

Finkler points out that the year 1510 is of especial importance in the history of influenza, as we here meet with the pestilence for the first time as a pandemic disease. It overran all Europe. Its historian, Dr. Thomas

Short, thus describes the outbreak:—"The disease called *Coccoluche*, or Coccolucio (because the sick wore a cap or covering close all over their heads), came from the island *Melite* in *Africa*, into *Sicily;* so into *Spain* and *Italy*, from that over the *Alps* into *Portugal*, *Hungary*, and a great part of *Germany*, even to the *Baltic Sea;* every month shifting its situation with the wind from East to West, so into *France*, *Britain*, etc., *Valeriola*, *Pechlin*, etc. It attacked at once, and raged all over *Europe*, not missing a family and scarce a person. A grievous pain of the head, heaviness, difficulty of breathing, hoarseness, loss of strength and appetite, restlessness, watchings, from a terrible tearing cough. Presently succeeded a chillness, and so a violent cough, that many were in danger of suffocation. The first days it was without spitting; but about the seventh or eighth day much viscid phlegm was spit up. Others (though fewer) spit only water and froth. When they began to spit, cough and shortness of breath were easier. None died except some children. In some, it went off with a looseness; in others, by sweating. Bleeding and purging did hurt. . . . Where blood was let, the disease proved malignant and pestilential, being attended with a violent, cruel, and unheard-of malignity, and made bad work."

The reader will admit that the foregoing description is a faithful portraiture of the influenza of our own day.

From time to time during the succeeding centuries epidemics of the disease occurred. An outbreak in the spring of 1743 was well described by J. Huxham, who adopted the name "influenza" for the complaint in the sentence—"This fever seemed to have been exactly the same with that which, in the spring, was rife all over Europe, termed the 'influenza.'" It is noteworthy that the French name for the malady, "la grippe," came into use also in connection with the outbreak of 1743. Biermer writes: "In France the 'influenza' has generally been called la grippe since the epidemic of 1743. This designation is probably derived from *agripper* (to attack), and not likely from the Polish word *chrypka* (*raucedo*), as T. Frank believes." In September 1758 influenza became epidemic in Edinburgh and in Scotland at large. Dr. Robert White, F.R.S., Professor of Medicine in the University of Edinburgh, the historian of this outbreak, observes that early in September, before the influenza appeared, a disease existed among horses in Perthshire—"the horses were observed to be more than usually affected with a cold and a cough." This is the first mention of the coincidence of an epizootic with influenza. In 1775 again Dr. Thomas Glass of Exeter notes that in his part of the country, in September, "many horses and dogs were severely afflicted with colds and coughs." Dr. Fothergill, in his *Sketch of the Epidemic Disease which appeared in London towards the end of the year* 1775, says that "during this time horses and dogs were much affected—those especially that were well kept. The horses had severe coughs, were hot, forbore eating, and were long in recovering. Not many of them died that I heard of, but several dogs."

Coming down to our own time, the occurrence of the so-called "pink-eye" among horses has been frequently observed since the great pandemic outburst of influenza in the autumn and winter of 1889. In his work on *Horses and Stables*, Lieut.-General Sir F. Fitzwygram, Bart., says: "The term 'pink eye' has of late years been applied to a particular form of influenza manifested by symptoms of a peculiarly marked nature, notably that of a remarkably clear pink coloured condition of the conjunctival membrane accompanied by a swollen or œdematous state of the conjunctivæ." He adds that "pink eye" is now generally recognised as being merely a modification of the catarrhal form of influenza. It may be remarked that in "pink eye" there is a noticeable tendency towards the formation of fibrinous clots or thrombi in the cavities of the heart and in the larger arteries, especially in the pulmonary system—a condition which not infrequently leads to unexpected and even sudden death.

In his Report to the Local Government Board (England) on the great epidemic of 1889-90, Dr. H. F. Parsons points out that, prior to the outbreak of influenza in London and some other parts of England, and also in some continental countries, a disease termed "influenza" prevailed among horses. In certain large stables in London numerous horses were affected by this epizootic in October 1889, some six weeks before the human epidemic made its appearance. Dr. E. Symes Thompson, the author of *Influenza, or Epidemic Catarrhal Fever*, says that he was so strongly impressed with the intimate connection between "pink eye" in horses and human influenza, that in the early part of December 1889 he wrote to the *British Medical Journal*, calling attention to the prevalence of an equine epizootic, and suggesting that it would not improbably prove the forerunner of an outbreak in man. These facts appear to establish a relationship between human and equine influenza, but a closer examination throws considerable doubt on such a relationship. In the first place, human influenza is reproduced in animals by inoculation only with difficulty and most inconstantly. Bollinger of Munich also believes that equine influenza and human influenza are essentially distinct diseases, because in the great horse epizootic resembling influenza which overran North America in 1872-73, there was no corresponding epidemic among human beings. Again, in the outbreak of 1889-1890, persons having to do with horses were not observed to be specially or earliest affected, while influenza prevailed epidemically at certain places, such as Newmarket, where no similar disease among horses was affirmed. Lastly, the bacteriology of the two diseases differs. Pfeiffer's bacillus is wanting in horse influenza, while Schutz, in 1888, cultivated a streptococcus from the lymphatic glands of horses suffering from "epizootic influenza," known in Germany as *Druse des Pferdes*, which he believes to be the specific infectious agent in this disease, and to which the name of *Streptococcus coryzæ contagiosæ equorum* has been given.

After the epidemic of 1758 the chief outbreaks of influenza in the eighteenth century were in America in 1761, Europe in 1762, Asia and Europe in 1781-82—"one of the most widespread pandemics of this disease that have ever occurred," according to Finkler; Europe in 1788-89, and America in 1789.

As pandemics of influenza in the nineteenth century, Finkler quotes the outbreaks of 1802-1803, 1830-33, 1836-37, and 1847-48, as being generally recognised. Hirsch adds to this list the epidemics of 1850-51, 1855, 1857-58, and 1874-75. Lastly, comes the great pandemic of 1889-90, with its repeated "flarings" during the succeeding decade—flarings which have not yet ceased to appear.

The history of this notable outbreak begins in May 1889, when influenza appeared in Bokhara, in Central Asia, thence travelling north-eastwards to Tomsk in Siberia, north-westwards to St. Petersburg, and westwards to the Caucasus and Southern Russia. In the last quarter of the year named the epidemic spread rapidly from Eastern Europe to the central and western countries. Early in 1890 it extended to America, Africa, and Australasia, "so that," says Finkler, "we may truthfully designate its appearance in 1889-90 as pandemic." "The high flood of the pandemic," observes the same writer, "flowed over the whole globe in the space of a few months."

As it travelled westward the disease was known by some name connecting it with the country whence it spread. Thus, in Siberia it was the "Chinese distemper;" in Russia, it became "Siberian fever;" in Western Europe it was the "Russian influenza."

ETIOLOGY

A theory was propounded in 1890 by the Hon. Rollo Russell that the pandemic of 1889-90 might possibly be traced to the diffusion of atmospheric dust in the wake of a great flood which had occurred early in 1888, in that part of China through which the Hoang Ho, or Yellow River, flows. This theory is everywhere discredited, although Dr. E. Symes Thompson in 1890 threw out the suggestion that dust in the air, whether Chinese dust or meteoric dust, might supply the "raft" for the aerial conveyance of the microbe of influenza. The presence and diffusion of dust in the air, according to Thompson, might be the condition which determined the prevalence of the disease, and might be adequate to convert a local epidemic into a universally distributed or pandemic wave. In the *British Medical Journal* of May 9, 1891, the present writer offered a meteorological explanation of the transference of influenza from point to point on the earth's surface.

Influenza had been raging in North China since the beginning of the winter of 1890-91. It appeared in several towns in the United States of America early in March 1891, and spread eastwards from Chicago, Pittsburg, Cleveland, and several other places in Ohio and in Iowa, finally invading New York and other cities near the eastern sea-board. About the second week in April an outbreak occurred in Norway, 891 cases being reported in one week in Christiania, and also in Sweden, notably in Göteborg. Almost simultaneously a severe outbreak took place in Yorkshire and Lincolnshire, extending thence southwards and westwards to the Midlands and the south-eastern counties of England.

The presumption is that the influenza microbes were wafted from China across the Pacific to North America by the air-currents connected with a winter barometric depression lying over the North Pacific. Subsequently the germs would be distributed along the eastern sea-board of the North American continent by the north-westerly winds of the American winter anticyclone or area of high atmospheric pressure, which results from the piling up of masses of cold, dense air over the land in winter.

Proceeding on the lines of the same hypothesis, the agency by which the influenza outbreak in North-Western Europe was produced was a spring anticyclone over Scandinavia and that part of the North Atlantic to which the name of the "Norwegian Sea" has been given. Reference to the weather charts published in the *Daily Weather Report* of the Meteorological Office, London, will show that almost uninterruptedly from the 1st to the 25th of April 1891 an anticyclone hung over either Scandinavia or the Norwegian Sea, or over both these districts. Under such circumstances, easterly and north-easterly winds—descending from the higher strata of the air, and hence so keen and dry as these winds are known to be—played upon the very districts in Sweden and Norway, and England, which were simultaneously affected with epidemic influenza.

In his Report to the Local Government Board for England and Wales on the *Epidemic Influenza of* 1889-90, Dr. H. Franklin Parsons observes that the progress of the epidemic in a direction from east to west, *i.e.* in a direction contrary to the winds prevailing at the surface of the ground, is at first sight opposed to the hypothesis of an infective material being carried by the air. Dr. Alexander Buchan, LL.D., Fellow of the Scottish Meteorological Society, explains this progress by supposing the material to be caught up by the ascending currents into the upper regions of the air, where currents prevail in different directions from those at the surface of the earth, and brought down again in other places where descending or anticyclonic currents exist. The late Rev. Clement Ley, Inspector of the Meteorological Office, London, however, informed Dr. Parsons that the atmospheric conditions prevailing during winter over the central and northern regions of the continents of Asia and Europe are such that upward currents cannot occur there at that season—the cold which prevails over the great continental tract in question renders it throughout the winter an area of high barometric pressure and descending air-currents. This reclamation, nevertheless, does not affect the hypothesis given above as to the aerial transference in the winter of 1890-91 of influenza germs from China to North America, and thence to Scandinavia and the British Isles.

The mode of spreading and the prevalence of influenza are apparently quite independent of season, and weather, and climate—a circumstance which distinguishes influenza from epidemic bronchial catarrh. Petrus Salius Diversus wrote—“Et tempore frigidiori et calidiori, et flante tam Austro quam Borea et pluvioso et sereno cœlo, peragravit hasce omnes Europæ regiones, et omnia loca indiscriminatim.”

Nor do overcrowding, want of ventilation, and other sanitary shortcomings seem to favour the spread of this disease. In the epidemic of 1889-90, as observed in Dublin, influenza spread as early and as quickly through the suburbs as it did through the crowded streets and alleys of the city. This is in marked contrast to the behaviour of such infective diseases as typhus, small-pox, scarlatina, and measles.

Several writers advance the view that influenza is not so much a miasmatic as a miasmatic-contagious disease. Thus, Dr. P. Duflocq, chef de clinique in the Faculty of Medicine in Paris, concludes an elaborate article on the clinical varieties of influenza observed in that city in December 1889 and January 1890, with these words:—“La grippe semble donc être une maladie à la fois épidémique et contagieuse, et la période d’incubation serait de deux jours.” According to this view the influenza germs produce their toxic effect chiefly by direct transmission, but also through the air outside of the human body.

A third school regards the disease as essentially and exclusively contagious, that is, transmissible from the sick to the healthy just as typhus, or scarlatina, or measles, or small-pox is. According to this doctrine influenza spreads from person to person directly or indirectly. The most usual mode of infection is by direct contact with those ill of influenza. But there is some evidence to show that healthy persons who have been in attendance on influenza patients may carry the infection to others while remaining well themselves. And, again, instances are recorded in which the disease has been spread through the agency of inanimate objects such as baggage, furniture, and even food-stuffs. Bäumler, for example, says that the first case of influenza in Basle occurred in a person who had been engaged in unpacking a bale of merchandise, which had recently arrived from the greatly infected Magazine du Louvre in Paris. Finkler, who quotes this observation, also narrates the following incident:—“An officer of the ship *La Bretagne*, which was anchored in December 1889 in the harbour at Brest, became ill at his home in Brest on 11th December, three days, it is said, after the arrival of a number of packages from Paris, which were first unpacked by the officer. These packages then carried the disease to his ship, while the vessels *Borda* and *Austerlitz*, which were anchored beside this vessel, are said to have escaped the infection."

So long as the miasmatic or pandemic view of the origin of influenza was entertained, there was no need to seek for a period of incubation, the virus being supposed to be already "hatched" at the time of its reception into the human system—that is, at the time of infection. It was indeed even then observed that in most cases there was an appreciable interval between the reception of the poison and the development of the symptoms, the most common duration of this interval being one to three days. We now know that this is a true period of incubation. In the last decade a vast amount of evidence has accumulated as to the communicability of the disease, and the latent or incubative period has been determined at two days on the average. Dr. Dawson Williams considers that the usual duration of this period is probably two or three days. This agrees with Dr. Parsons' estimate exactly. Sometimes the symptoms of an attack seem to develop almost simultaneously with exposure to infection. In one instance, observed by the writer, a lady visited a friend ill of influenza at 2 P.M. Three hours later she was attacked with symptoms of the disease—chills, weakness, coryza, lacrymation, stuffing of the nostrils, loss of smell and of taste. Dr. George Neale of Cadoxton, Glamorgan, in his own case, had a rigor an hour after visiting his first influenza patient. On the other hand, the period of incubation has been prolonged to several days, although a duration of latency exceeding seven days must be regarded as most exceptional.

In considering the effect of an epidemic of influenza upon the public health, we are, unfortunately, at a serious disadvantage in having no system of registration of disease in the United Kingdom. Experience of recent outbreaks, however, shows that, with the coming of influenza, the whole "epidemic constitution"—to use Thomas Sydenham's classic phrase—changes rapidly for the worse. The power of resisting disease is lessened among a people smitten with this plague, and extreme languor with prostration passes over the population like a destructive tidal wave.

The writer's observations on the epidemic of 1889-90 in Dublin extended over a period of nearly three months, from early in December 1889 up to the end of February 1890. The two earliest cases which came under his notice, dated from Thursday and Friday, 5th and 6th December. One of these two patients consulted him on Monday, 9th December, having been ill from Thursday the 5th. He was a clergyman, aged fifty-two, in whose case the prominent symptoms were headache, tender eyes, palpitation, insomnia, and loss of strength. The outbreak was at its height in the first half of the following January. Towards the close of that month its epidemic prevalence waned quickly, but in the middle of February a recrudescence occurred. The following conclusions are justified by the facts:—

1. The epidemic of influenza was more pernicious to the population of Dublin than the extreme cold of January 1881.

2. It slew its victims, not so much directly, as by means of complications and sequelæ, affecting the breathing organs and the heart.

3. It spared the lives of children of tender years, but killed large numbers of adults and those advanced in life.

4. Its effect upon the death-rate was sudden and pronounced, and lasted for at least seven weeks, or throughout the month of January and first half of February.

The virus or contagium of influenza, when once introduced into the body, acts primarily and quickly on the nervous system, producing the phenomena of an acute pyrexia, with singularly rapid pulse. In January 1890 a boy of seven and a half years had incessant nausea and vomiting for 24 hours, and profuse sweating, with a pulse as fast as in scarlet fever —140 per minute. He was one of a family of five children, all of whom had influenza together, their ages ranging from thirteen to four years. The mildest cases were those of the eldest and youngest. There was a bronchial catarrh in two instances. Very young children seem to enjoy a certain immunity from the disease, or to have it in a mild form—an ephemeral fever, followed by profuse sweating, and after a few days a tendency to slight catarrh. Hilton Fagge says that influenza "is sometimes dangerous to very young children," but he adds in a footnote that "Sir George Baker writes of the epidemic in 1762, *Leviter plectebantur infantes et liberabantur facillime.*" This is altogether the writer's experience also. In 1847 Dr. Fleetwood Churchill communicated to the *Dublin Journal of Medical Science* observations he had made on the epidemic influenza observed in January and February of that year among children in Dublin. In upwards of sixty cases, embracing children of all ages, from two months old to twelve or fourteen years, he had not a single death; so that he was led to conclude that, among children, although the epidemic was very general and severe, yet the danger was not very great when the disease was properly handled. This "child-type" of influenza presented itself to the writer's notice in 1890 in a large girls' school, of which he is one of the physicians.

Clinical Features

Adults in most instances suffer much more severely than children from the influenza poison. In them the attack as a rule comes on with lightning-like speed. A person apparently in perfect health is suddenly overcome by a feeling of general discomfort and profound depression. He feels chilly, or is shaken with a rigor worthy of an ague fit. His head aches. There are pains in his eyeballs, or "behind the eyes," and the eyeballs are exquisitely tender on pressure. Soon rheumatoid pains rack his body. They are particularly severe in the nape of the neck, small of the back, in the knees, and along the margins of the ribs. The patient is often sleepless, sometimes delirious. He may temporarily suffer loss of the special senses of smell, taste, and sometimes hearing. His eyes smart, tears overflow, he shrinks from the light (photophobia). Occasionally there is intense earache (otalgia). Strength ebbs away far more quickly than even in typhus, so that after a few hours he must take to his bed. Nor do the digestive and respiratory systems escape. The tongue becomes thickly coated with a creamy or blanket-like fur. There is a bad taste in the mouth, the breath is heavy or even fœtid. There is complete loss of appetite, amounting to a loathing against food. Nausea and perhaps vomiting add to the sufferer's distress. The bowels are constipated, but occasionally diarrhœa sets in as in an attack of cholerine. In another group of cases the brunt of the poison appears to fall on the respiratory tract. Cough, expectoration, a sense of oppression on the chest, a tightness in the breathing being common symptoms. In a word influenza seems to have the property of picking out the weak point in a person's constitution. If the patient is neurotic, nervous and neuralgic symptoms are likely to develop. Any old tendency to catarrh of either the respiratory or the digestive mucous membrane is at once intensified in the presence or in the wake of this strange malady. At the same time catarrhal symptoms may be badly marked or entirely absent. Dr. Parsons, in his Report on Epidemic Influenza in 1889-90, states that the comparative absence of catarrh led some observers to doubt whether that visitation had been one of true influenza. But a similar infrequency of catarrh had been observed on previous occasions. Dr. Parsons quotes the following authorities. Sir William Gairdner, writing of an outbreak which occurred in 1862, says: "But although catarrh is frequent and may be severe, the disease is essentially a fever and not a catarrh. Nay, the catarrh may be absent or insignificant; not unfrequently it is so." Sir Samuel Wilks also, writing of the epidemic of 1847, says: "Although a synonym for influenza is 'epidemic catarrh,' the latter was by no means a constant symptom; many of the worst cases, and especially the fatal ones, having no catarrhal symptoms whatever." In a report on the epidemic of 1782, the Royal College of Physicians of London state that "the symptom which universally prevailed, and which appeared to be almost a pathognomonic of the disease, was a distressing pain and sense of constriction in the forehead, temples, and sometimes in the whole face, accompanied with a sense of soreness about the cheek-bones under the muscles. This, now and then, was felt previously to the catarrh, and not unfrequently was followed by very little or no catarrhous affection."

TYPES

The protean forms which influenza presents from a clinical aspect arise from the peculiar aptitude its poison has for fastening on the points of least resistance in the constitution. Of the many types described, perhaps the four following stand out most prominently:—

1. The neurotic, neuralgic, or rheumatic type.
2. The cardio-pulmonary type.
3. The gastric or gastro-intestinal type.
4. The febrile type.

It is proposed to give a brief clinical sketch of four cases, each illustrating a type observed in the epidemic of 1889-90 in Dublin.

I. *The Neurotic Type.*—One of the earliest cases which the writer saw in the epidemic of 1889-90 was that of a lady, who was seized on the evening of Friday, December 20th, 1889, and who was visited next morning by Dr. James Craig, one of the physicians to the Meath Hospital, Dublin. The following is this lady's own account of her attack:—"Friday, 20th December 1889, I went to the oratorio at St. Patrick's Cathedral apparently in my usual health. Shortly after entering the Cathedral I felt chilled, as if cold water was being poured down my back and legs. When I returned home I warmed myself at a good fire, was given some hot wine and water, and went to bed; then my face and head got very hot and uncomfortable, and pains began in my arms, shoulders, and legs. All night the pains were very bad, sometimes so sharp across the back of my chest that I could have cried out; and, although I felt burning to the touch, the cold-water sensation continued. I got no sleep that night. Next day, about twelve o'clock (mid-day), I was given a powder (salicylate of sodium), and in two hours afterwards another, which put me into a perspiration. The pains in my limbs got better, but my head began to ache badly, and all day I felt very ill. I suffered from great thirst. Saturday night slept better. Sunday morning about 5 A.M. I wished for a cup of tea, but could not taste it. I might have been drinking hot water. Sunday evening pains had quite gone. I had no headache. I got up for a while, but felt very weak. For several days I had no energy for anything, the least exertion tired me. My sense of taste did not return for four or five days. I also got a cough which was very troublesome. Temperature—Friday night 101°; Saturday morning 100°, evening 98·8°."

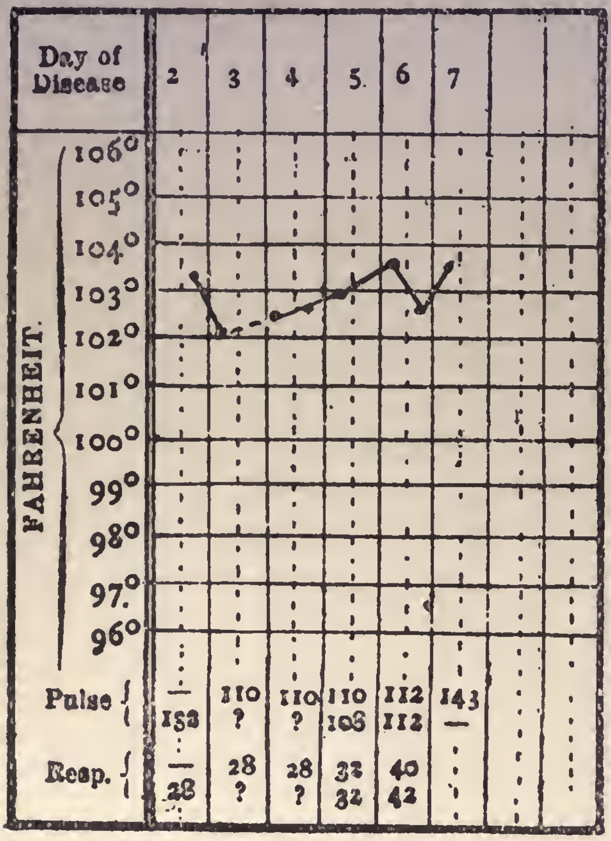


Chart I.—Mrs. W., aged 54.

II. *The Cardio-pulmonary Type.*—On Monday, December 30th, 1889, Mrs. W., a lady, aged fifty-four years, somewhat frail and delicate, while out walking was seized with shivering and violent headache, and intense pain in the back and "bones." On reaching home she at once went to bed, feeling very ill and prostrate. Next day the tongue was thickly furred and dry. Her pulse was 132; respirations, 28; temperature, 103·3°. Having regard to the sudden onset of the illness and the symptoms, the attack was declared to be one of influenza.

On New Year's Day (third day) the pulse was 110; respirations, 28; temperature, 102·0°. The tongue thickly coated; eyes tender and lacrymation; complete anorexia; great prostration.

January 2, 1890 (fourth day).—The report was that she had a better night. Herpes was showing round the nostrils. Pulse, 96-100; respirations, 28; temperature, 102·4°. Severe stabbing or catching pain was complained of at the lower part of the left side of the chest. No physical signs could be detected, and a hot poultice relieved the pain.

January 3 (fifth day).—Pulse, 110; respirations, 32; temperature, 102·7°. A lymphy crepitation was now audible over the upper part of the left side of the chest, and on deep inspiration a fine pneumonic crepitation could be heard.

January 4 (sixth day).—Pulse, 110; respirations, 40; temperature, 103·2°. Dulness now existed, which was rapidly extending all over the left apex, where also a marked *frottement* could easily be recognised. At 6 P.M. Dr. Watson Pike, R.A.M.C., saw the patient in consultation and agreed in the diagnosis of influenza complicated with a left pleuro-pneumonia. Pulse, 112; respirations, 42; temperature, 102·7°. There was not a trace of expectoration, and scarcely any cough occurred. We considered the patient to be in danger, and continued the treatment, which consisted in free stimulation, frequent feeding, and quinine.

At 1.30 A.M. of Sunday, January 5th, the patient was sinking fast. Her pulse was failing, and the temperature was 103·3°. She rallied for a time, but at 6 A.M. another attack of cardiac failure came on. From this also she rallied, but at 10 A.M. she suddenly died.

As bearing on the diagnosis of this case, it is to be noticed that four—if not five—of the other members of this lady's family suffered from influenza either immediately before or after her illness.

Appended is the clinical chart.

The foregoing case illustrates the fact that influenza, while infrequently directly fatal, causes an indirect loss of life which is appalling, chiefly through complications affecting the respiratory, and, in advanced life, the circulatory systems. It has been said that influenza, while relatively less fatal, is absolutely more fatal than cholera.

The writer has seen fatal cases of influenzal bronchitis, pneumonia, pleuritis, and heart failure. The pneumonia, while producing the ordinary physical signs of acute croupous pneumonia, is often latent in its course, or accompanied by a profuse muco-purulent expectoration, with scarcely any rusty sputa. The ebbing of the strength in some of these cases in elderly people is something awful—it is often absolutely beyond control.

Younger subjects have a better chance of "pulling through." For example, a young lady, aged nineteen, was suddenly attacked with shivering, headache, and high temperature, with very rapid pulse, on Friday, December 6th, 1889. The fever lasted a few days, and then gave way, to be followed by a second rigor on Friday, December 20th, and urgent chest symptoms. On visiting her with Dr. Usher of Dundrum on the 22nd, her pulse was 124, respirations were 36, and the temperature 102·7° at 3 P.M. The left base was solid. Great dyspnœa, tubular breathing, and marked bronchophony were present. She was bringing up an abundant muco-purulent sputum—like that of bronchiolitis rather than acute lobar pneumonia. It was examined by Dr. Bewley, who reported that no tubercle-bacilli were present, but that various putrefactive microbes were found. On December 26th, her pulse was 120, respirations had risen to 48, and the temperature at noon was 101·6°. Dr. James Little saw her with Dr. Usher and me next day, when we found a new patch of pneumonic consolidation in the middle of the right back, and there were a few rusty-tinged sputa. From this time she steadily recovered, and went to Bournemouth with her father on January 23rd, 1890, "pretty nearly all right," as he expressed it in a letter to me.

III. *The Gastric Type.*—On Wednesday, January 8th, 1890, Mr. W. B. S. enjoyed a day's shooting in the County Wicklow. The following day he returned to town in his usual good health; but in the afternoon felt chilly, complained of headache and nausea, and felt utterly miserable. He went to bed early, but passed a wretched night—restless and sleepless. Next morning he complained of pains in the eyeballs, back of the head, and small of the back. Pulse, 84; temperature, 99·9°; tongue thickly coated; complete loss of appetite and nausea. He felt entirely prostrate, and expressed his belief that some fish which he had eaten for dinner had thoroughly disagreed with him. Two miserable days of sickness followed, the temperature rising on the morning of the 5th day to 103·1°. A short cough had set in, and the eyes were suffused and tender. There was constipation, and he complained of weight and fulness in the pit of the stomach. Dr. James Little saw him with me and thought it likely that the fever would run on for some time. A quiet day gave promise of a restful night, and this promise was abundantly fulfilled. He had an excellent night, partly due to 20 grains of phenazone, with 20 minims of tincture of gelsemium in a draught in four divided doses

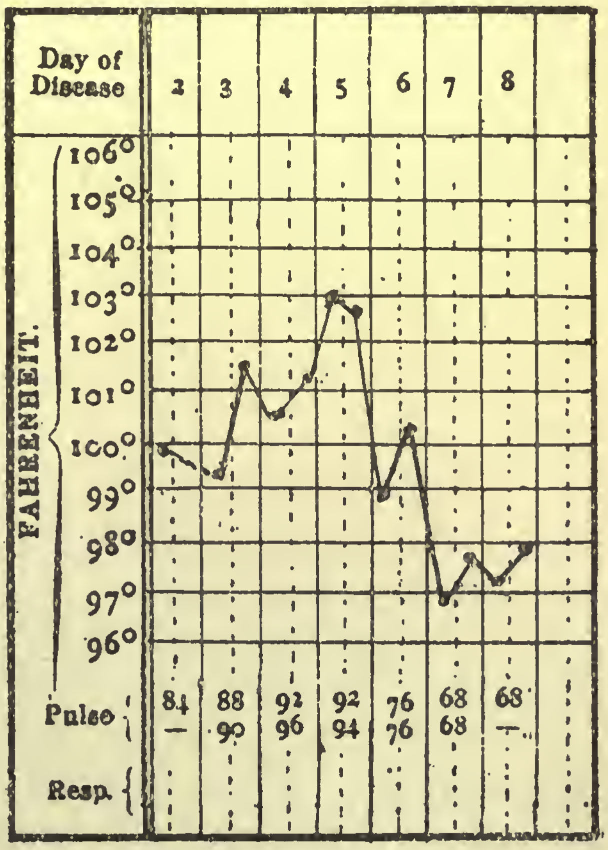


CHART II.—Mr. W. B. S.

at bedtime. Next morning, pulse 76; temperature 98·7°, rising to 100·2° in the evening, but without any return of restlessness. Subnormal temperatures followed for a few days—96·4° being one observation. The tongue cleaned very slowly, and several days of extreme languor and weakness preceded final convalescence.

This gentleman's wife had, a few days previously, suffered from influenza, from which she was recovering when he fell ill. She nursed him and got a relapse, accompanied with cough, bronchial catarrh, and absolute loss of appetite. It is right to mention that Mr. S. had been subject to occasional "bilious attacks," as he described them. Except for these he had not known a day's sickness from his boyhood, and did not recollect when he had been obliged to keep his bed even for a day.

IV. *The Febrile Type.*—On New Year's Eve 1889 I received a note from a surgical colleague asking me to see him, as he had been taken ill the same afternoon while in his study. On visiting him in bed, he was still shivering at times, and complaining of a distressing feeling of cold water streaming down his back. He stated that he had been out of sorts for three or four weeks, and it was quite evident that he had made up his mind that the attack was one of typhoid fever. His pulse was 92, the temperature about 100°. His tongue was very furred, and his eyes were injected with swollen lids. He had lost the senses of taste and smell, and complained much of rheumatoid or neuralgic pains in the back and limbs. Two restless feverish nights followed. So I asked our mutual friend, the late Dr. Samuel Gordon, to see him with me. At the end of the examination the patient said to Dr. Gordon, "Our friend Moore says I have influenza; do you think I have?" The answer was, "I do not think it, but I am sure of it."

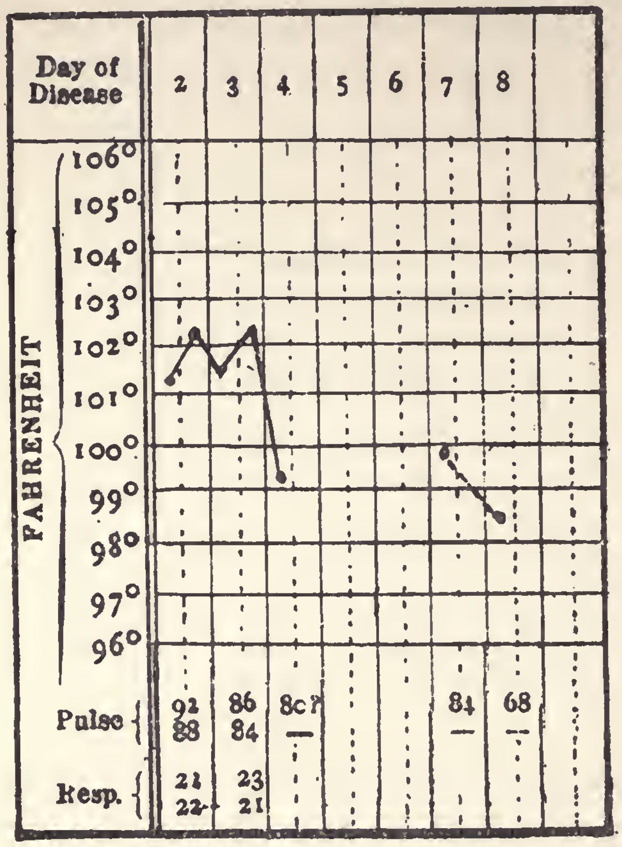


CHART III.—W. S.

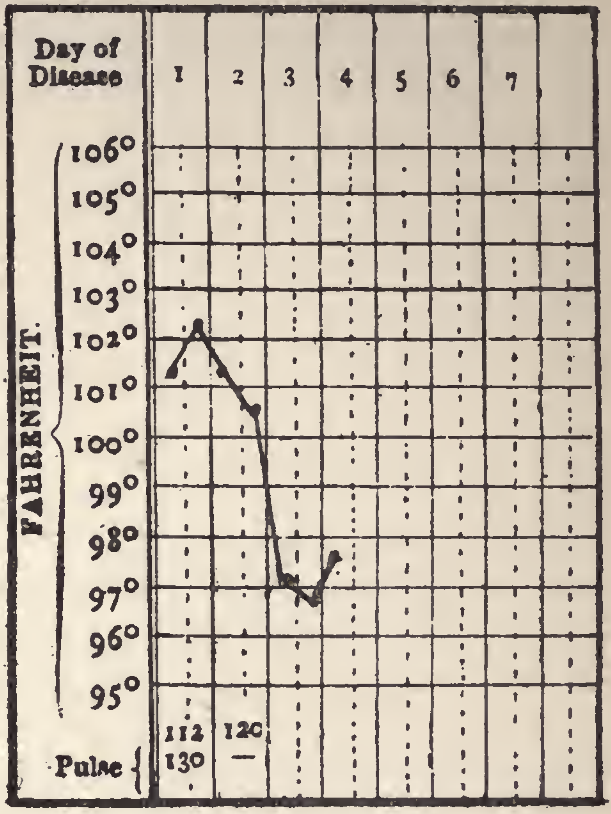


CHART IV.—Elsie M.

There was in this case steady pyrexia for four days, as shown in Chart III., and then came profuse sweatings, lasting for several days. A slight elevation of temperature occurred on the evening of the seventh day, after which convalescence went on uninterruptedly. The weakness was for a time extreme.

This febrile type prevailed especially among children. The three charts, numbered IV., V., VI., show the febrile movement in girls, aged from 12 to 15. The marked subnormal temperatures on the third and following days are very noteworthy, and are so constantly present in the defervescing stage of influenza as to become an important diagnostic sign.

The febrile movement in even uncomplicated influenza is, as Wunderlich would say, "polytypical," or "atypical." This is shown by the charts already given, and, further, in a diagram containing three different types of temperature range in influenza (which is reproduced), illustrative of a paper, "Zur Kenntnis des Fieberganges bei Influenza," by Dr. Otto Frentzel, assistant physician to the Municipal General Hospital at Friedrichshain, which appeared in the *Centralblatt für klinische Medicin* for January 11th, 1890.

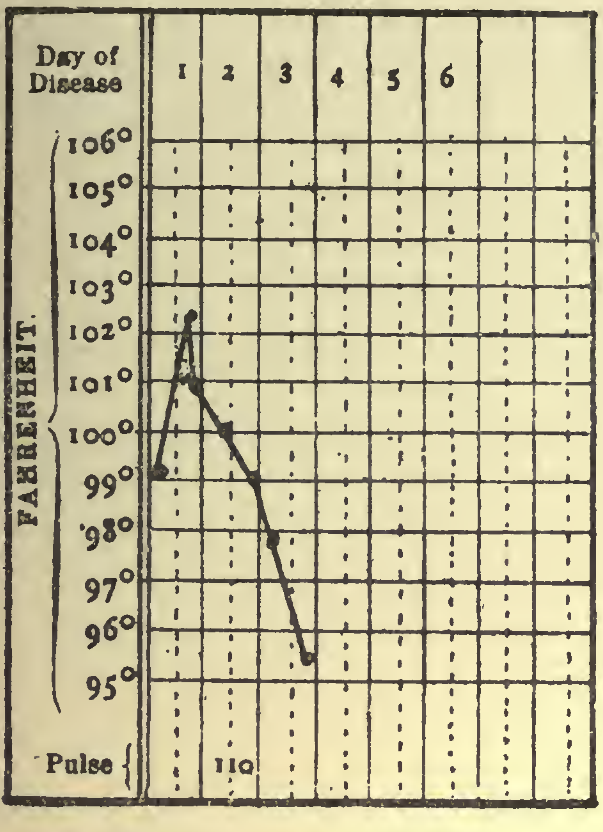


Chart V.—Dorothy O.

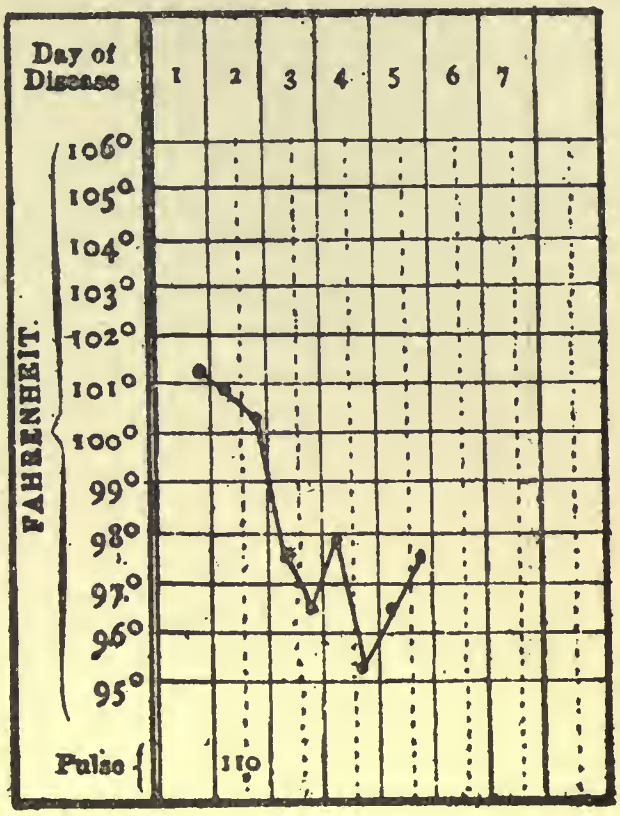
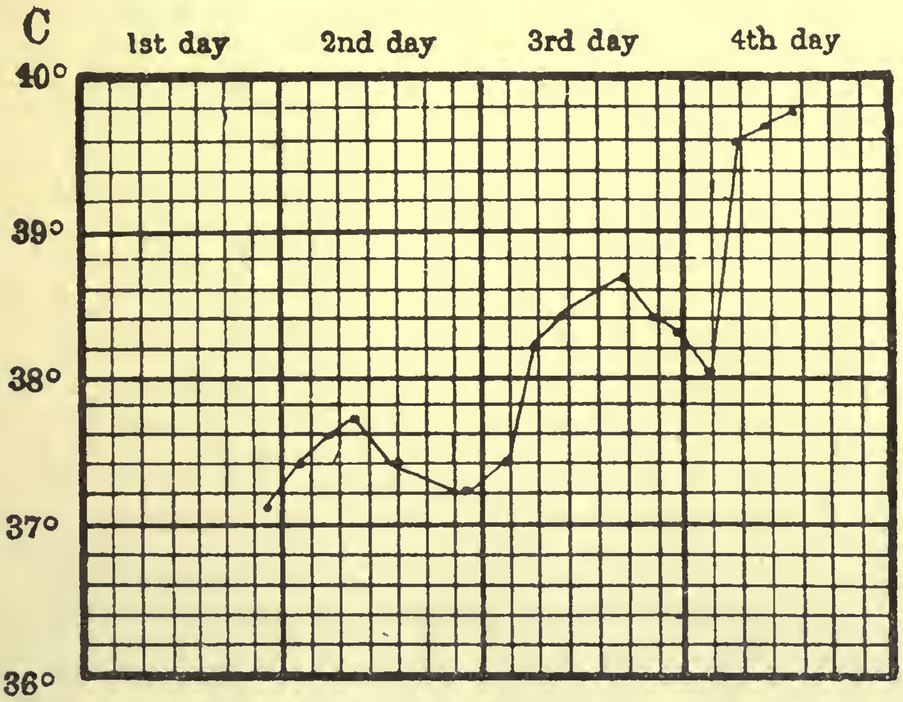


Chart VI.—Fanny C.

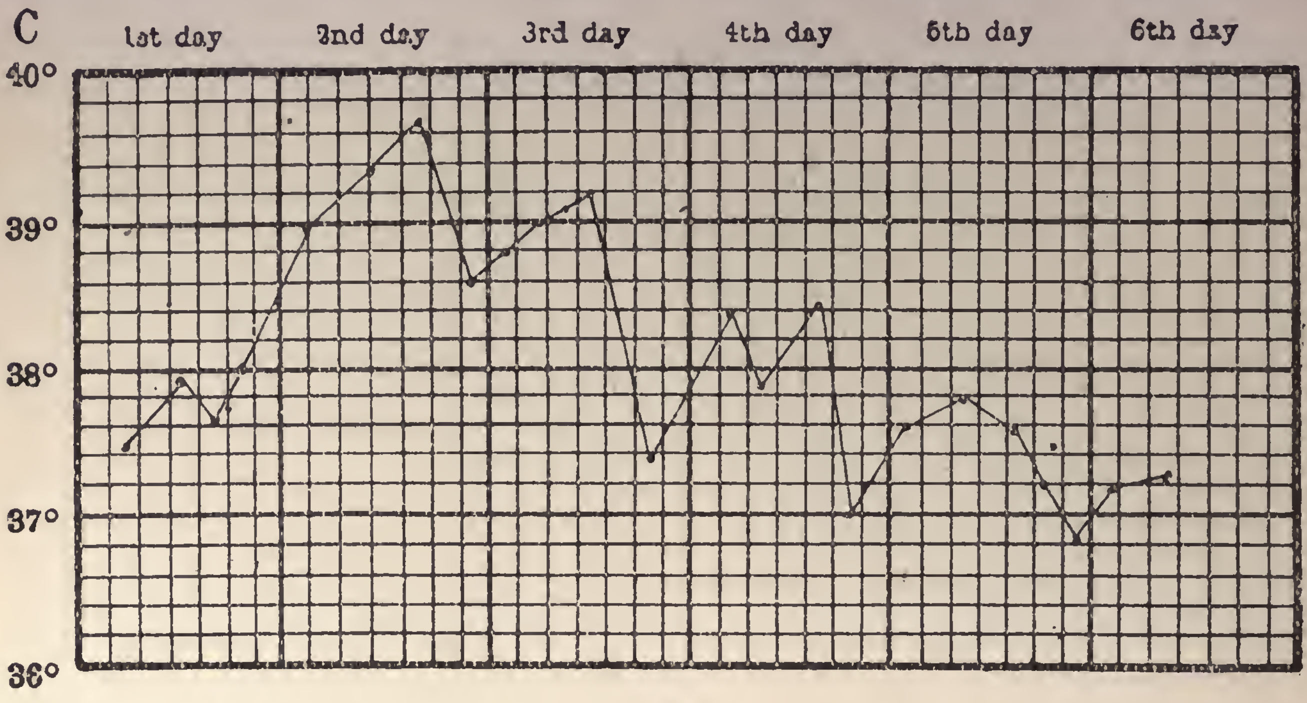


Type I.

Influenza shows a marked tendency to relapse, and to this feature the indirect fatality of the malady is largely due. In the epidemic of 1847 the death-rate was estimated at only 2 per cent of the cases observed in

London. It was probably not higher in the several epidemics which have occurred since 1889 up to the present date (1900). In the winter of 1889 at least a fortnight elapsed between the occurrence of the earliest cases and the fatalities from influenzal bronchitis and pneumonia.

So far from establishing immunity, an attack of influenza seems to predispose an individual to a second or even a third visitation of the disease. In this respect influenza resembles erysipelas, diphtheria, and perhaps pneumonia, or "pneumonic fever."

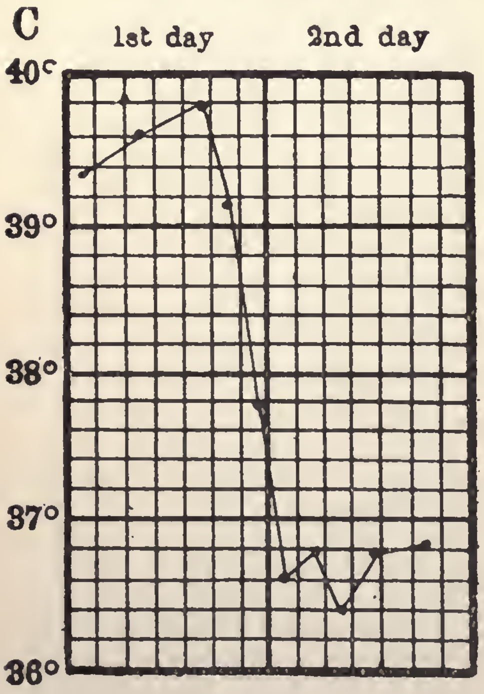


Type II.

Atypical Forms

Apart from the very many cases which range themselves more or less accurately under the four types just described, there remain not a few cases of influenza which do not admit of being properly classified. They are atypical—that is to say, they conform to no type. With equal propriety such cases might perhaps be called polytypical, or many-typed.

In the first place, as Zülzer pointed out in his classical monograph on "Influenza" in von Ziemssen's *Cyclopædia of the Practice of Medicine* (1875), a great number of rudimentary forms are observed in every epidemic. "A considerable part of the population in fact, under the influence of the 'genius epidemicus,' exhibits a state of indisposition which does not amount to a full febrile affection, but which is shown to be a general invasion of the system by slight coryza, by confusion of the head, by one's quickly becoming fatigued, by disinclination for business, and often by sore throat, tickling cough, etc." Zülzer further shows that it is seldom possible to bring all the special cases of influenza within the compass of the ordinary classification because the forms under which the disease manifests itself are made up of various combinations.



Type III.

Lastly, the same author observes that the "features of the disease acquire still greater diversity from the various *complications*." Many phenomena of the disease, especially the nervous symptoms, exhibit "important modifications according as they affect more or less irritable individuals; in hysterical and such other patients influenza often assumes a pronounced nervous or spasmodic character. In the case of hæmorrhoidal affections, and in the rheumatic or gouty diathesis especially, the muscular pains will occur. With young children symptoms of congestion of the brain are often seen, and this perhaps may be the explanation of the unfavourable result sometimes observed in their cases."

Perhaps also the form of the disease which Finkler has called "chronic influenza" should be included among the atypical or aberrant varieties. When Finkler asserts that influenza may assume a chronic character, he refers to the actual continuance of the influenza bacilli and to a resulting chronic bronchitis or chronic pneumonia. Various authors, among them Pfeiffer, Wassermann, Beck, and Kruse, have assured us that the bacilli of influenza may repeatedly be found in the sputum for weeks and months. In such patients cough and expectoration persist after the influenza has seemingly passed away; and, without any known cause, at intervals of a few days or a few weeks, the symptoms of a bronchial catarrh return, so that again and again cough and expectoration increase—to decrease in turn, probably without medical attendance or treatment. In these chronic cases of influenza, Finkler says he is prepared for everything which occurs in the acute form—sudden fever, neuralgia, attacks of perspiration, digestive disturbance, asthmatic symptoms, coryza, sore throat,—all these reappear without warning, alternating in such a manner that the patient often attributes the cause to a "change in the weather." Only a bacteriological examination—which should always be made—will distinguish such cases from a bronchial or pulmonary tuberculosis, or from a secondary diplococcus infection.

Effects on different Systems

The statement made above that influenza seeks out the weak point (*locus minoris resistantiæ*) in an individual gives a clue to the multiplicity and severity of the complications and sequels of the disease.

The Respiratory Tract.—As was to be anticipated, the poison of influenza falls with special violence on the respiratory tract. In Dublin, during the first quarter of 1890, 973 deaths were referred to diseases of the respiratory system, exclusive of phthisis. This number was 34 per cent in excess of the average for the corresponding quarter of the previous ten years. The deaths from bronchitis were 654, against an average of 507, or an excess of 29 per cent. Those from pneumonia were 211, or 79 per cent above the average.

Foremost among the secondary affections which complicate influenza stands pneumonia, to which the heaviest death-toll is paid by the victims of the primary or influenzal infection. But, in this instance, the term pneumonia must be taken in a generic rather than in a specific sense. As a matter of fact, broncho-pneumonia, true lobar (croupous) pneumonia, and cellular (influenzal) pneumonia are well-recognised varieties of lung-inflammation which occur in or after influenza.

Leyden, in a communication to the Medical Society of Berlin, states that the pneumonias observed by him showed a peculiar course—severe pain in the side and dyspnœa were rarely noticed; the local process was not altogether typical; frequently it was necessary to watch for three or four days before any evidence of a localisation of the disease was forthcoming. Then a crepitating râle was heard over a wide area, and this perhaps the very next day would have disappeared to show itself in some other situation. Not very often a firm hepatisation occurred with clearly mapped out dulness. Again the typical sputum of pneumonia was often wanting. Bacteriological investigations revealed the presence of three kinds of microbes: (1) Diplococci, which represented the well-known pneumonia-diplococci of Fränkel; (2) Streptococci; (3) Staphylococci. Leyden adopted the view that the forms of pneumonia are different—typical genuine pneumonias with deviating course; mixed forms, especially in those combined with pleural effusion; lastly, simple streptococci pneumonias.

Ribbert, discussing the possibility of a causal significance of the *Streptococcus pyogenes* in relation to the phenomena of influenza, alludes especially to the inflammation of the lungs, whose peculiar erysipelas-like spread, on which Finkler lays so much stress, and whose anatomical relations admit of being referred back to the influence of the streptococcus. Ribbert points out that, in contrast to ordinary croupous pneumonia, the cut surface of the hepatised lower lobe in three cases presented an almost smooth appearance, the exudation was soft, very rich in cells, and poor in fibrin (hypinosis). Cultivation experiments with the tracheal mucus, the lung tissue, the spleen, and the kidneys, furnished in five out of eight cases the *Streptococcus pyogenes*, or else the *Streptococcus erysipelatosus* (which has been shown to be identical with the former), the presence of which microbe could be demonstrated in the sputum also of the influenza patients. His investigations on the whole yield the result that in all cases in which micro-organisms were at all capable of demonstration, the *Streptococcus pyogenes* was found. Only once was there in addition a coccus which had a great resemblance to the *Diplococcus pneumoniæ* and probably represented a modification of the same.

Finkler observed 45 cases of influenzal pneumonia, of which only 2 came under the description of typical lobar pneumonia, while the other 43 were regarded as cases of the disease which he has often described as "Streptococcus Pneumonia." Seven of his patients died, post-mortem examinations being made in three instances. He regarded the pathological condition as a preponderating cellular inflammation with participation of the interstitial tissue. The cellular nature of the inflammation, together with the pronounced tendency it exhibits to develop by spreading indefinitely, in Finkler's opinion justified him in describing this disease as an erysipelas of the lung. He points out that the resemblance of this form of pneumonia to erysipelas consists, not alone in the anatomical characters of the inflammatory process, but also in the fact that both diseases depend on the presence of streptococci. Finkler looks upon this streptococcus pneumonia as a localisation of the exciting cause of influenza in the lungs. As to this last point one would be more inclined to agree with Leyden and Levy that the question is much more one of a secondary infection, for which the influenza merely laid the foundation.

It is to be remembered that the foregoing observations were made in connection with the great pandemic of influenza in 1889-90, antecedent therefore to the discovery by Pfeiffer in 1892 of the *Bacillus influenzæ* in the purulent bronchial secretion, and by Canon in the blood of patients suffering from epidemic influenza. That discovery must certainly be regarded as conclusive proof of the existence of a specific primary infection of the lungs to which all other infections are accidental and secondary.

We may then conclude that Pfeiffer's bacillus itself produces a catarrhal bronchitis and a pneumonia, while the presence of influenza predisposes to secondary infections of the lungs by the *Diplococcus pneumoniæ* of Fränkel, the *Streptococcus erysipelatis* of Fehleisen, the *Bacillus pneumoniæ* of Friedländer, and possibly the *Staphylococcus pyogenes aureus* of Ogston.

The *pleura* is often involved in the primary or secondary pathogenic processes of influenza. Researches carried out by a Collective Investigation Committee in Germany showed that the pleura became engaged in from 12 to 33 per cent of the cases of pneumonia complicating influenza. At Riga Krannhals met with "exudative pleuritis" in 12 out of 220 cases of influenza. In the Riga hospital there were 7 cases of pleural effusion among 87 cases of influenza. The exudation was, as a rule, sero-fibrinous, but in 6 instances it was purulent. The affection may supervene upon bronchitis without pneumonia. The bacterial factor in its production is most frequently the *Streptococcus pyogenes*, but various staphylococci also play a prominent part in its etiology. Finkler of Bonn alludes to the intense dyspnœa which often attends the pleuritis of influenza—a dyspnœa which he rightly regards as not merely mechanical, but also probably toxæmic in its origin. Long ago Robert James Graves of Dublin gave a graphic description of the extraordinary dyspnœa witnessed in the influenza of 1836-37. According to him, "even in many cases where the bronchial mucous membrane is but slightly engaged, the amount of dyspnœa is remarkably great. Indeed, it might be said with much truth that the dyspnœa was by no means proportioned to the extent of pulmonary inflammation." Having pointed out that in many bad cases of influenza the dyspnœa is intermittent, Graves suggests that the respiratory derangement may depend on the same general cause which produces the whole train of symptoms, and that it might exist even where there was no bronchial inflammation at all. "The dyspnœa," he adds, "appears to be chiefly attributable to some impression made on the vital activity of the lung."

Influenza is a perilous complication of pulmonary consumption. In London at the height of the epidemic of 1889-90 the weekly number of deaths from phthisis rose to double the average. In Dublin phthisis caused 437 deaths in the first quarter of 1890, or an increase of 90 deaths, or 26 per cent, as compared with the average for the first quarter of the previous ten years.

So long ago as 1803 Dr. Woodford of Almsford wrote: "When the influenza attacked subjects apparently predisposed to phthisis, it never failed to increase and call into action the latent seeds of that disease, and in several instances to induce speedily a confirmed state of it, which quickly proved fatal." In this opinion most students of influenza will agree, although there have been a few dissentients from time to time. We are thoroughly in accord with Dr. J. F. Goodhart when he says that in a large number of cases influenza lays its victim open to an attack of pulmonary tuberculosis. "Many a case," he writes, "seemed to start from an attack of influenza, and many a case of phthisis was certainly sent on its way with an alarming increase in the rapidity of its progress."

Finkler has drawn attention to the fact that in phthisical patients particularly influenza lasts an extraordinarily long time, that it becomes as it were chronic in them, and from time to time induces fresh inflammatory processes in the cicatrising tuberculous areas in the lungs.

Partly also through the damage inflicted by it on the elasticity of the lung, but chiefly from its hurtful effects upon the pulmonary nerves, influenza lays the foundation for bronchial asthma, a sequel which is usually transitory, but may be persistent.

Another distressing sequel, to which Professor Osler has drawn attention, is diffuse bronchiectasis.

The Cardio-Vascular System.—Influenza *poisons* the heart. These words perhaps will best express the disastrous effect which this strange malady has upon the heart-muscle, but especially upon the innervation of the heart. Indeed, the term "heart failure," now on every one's lips, was scarcely ever heard before the pandemic of 1889-90. The very treacherous and dangerous form of influenza known as the cardiac type may occur with or without pneumonia. The frightful ebbing of strength in many cases of influenzal pneumonia in persons of middle life or advanced in years reaches its climax in a true heart-palsy. In other cases, uncomplicated with pneumonia, the heart collapses, sometimes with a suddenness that suggests rupture or cardiac thrombosis.

In Dublin the outbreak of 1889-90 had an immediate and untoward effect in raising the mortality from diseases of the organs of circulation, including "heart disease," in the most general sense of the term. A marked rise in the number of deaths coincided with the chief epidemic period, the second, third, and fourth weeks of 1890. In the fourth week diseases of the circulation were returned to the Registrar-General for Ireland as the cause of death in the Dublin registration area in 30 instances, compared with a ten years' average of 12·2 deaths for the corresponding week. Of the 30 victims, 14 were aged between 40 and 60 years, 9 between 60 and 80, and 4 were above 80 years.

In estimating the malign influence of the disease on this class of maladies, it is noteworthy that neither intense cold nor undue warmth in winter raises the death-toll from diseases of the circulatory system to any perceptible extent. This interesting fact throws into bolder relief the baneful results of influenza.

The grosser lesions of pericarditis and endocarditis must be accounted rare in or after influenza. Even evidences of myocarditis and acute degenerations of the cardiac ganglia and nerves are often sought for in vain, alike before and after death. Nevertheless, in a large number of cases the physical signs and symptoms bear testimony to a profound influence of the poison of the disease upon the myocardium and its nerves. Breathlessness on slightest exertion, coldness and blueness of the extremities, restlessness, the presence of blowing systolic murmurs at the apex, paroxysmal tachycardia, or arrhythmia of the pulse, epigastric pulsation—all point to acute dilatation of the ventricles—a pathological state brought about by changes (inflammatory or degenerative, or both) in the heart muscle and in the cardiac nerves. The pulse is not only rapid, irregular, or intermittent, but its rate varies remarkably with change of position, becoming extremely rapid when the patient sits up. This is what Huchard calls the *pouls instable.* The toxins of influenza may affect the blood-vessels, causing thrombosis or hæmorrhages, lesions which are strictly analogous to those which occur in the vessels in the course of other infective disorders. Nose-bleeding, hæmorrhage from the ear, hæmoptysis, hæmatemesis, bloody stools, hæmaturia, and menorrhagia have all been observed more or less often.

The Skin.—The skin may be the seat of various rashes in influenza. Thus in the hæmorrhagic variety of the disease petechiæ may occur. Or again, from vaso-motor paresis, fleeting erythematous rashes (erythema fugax) may develop, resembling either measles (morbilliform) or scarlatina (scarlatiniform). Papular sweat rashes, with miliary vesicles or sudamina, are also met with. Herpetic eruptions are especially common.

In contrast to dengue fever, however, influenza must be regarded as a non-eruptive fever. When the rashes just mentioned do appear, they are accidental rather than essential or specific, and they result from vaso-motor paresis, or hyperpyrexia, or profuse sweating, or from the ingestion of such drugs as quinine, phenazone and its congeners, or salicylate of sodium.

Erysipelas, eczema, acute furunculosis, and suppurative follicular impetigo have also been observed as complications or sequelæ. Pemphigus has occurred in rare instances.

Extreme dryness of the skin is a rare epiphenomenon. Extreme sweating (hyperidrosis) is not uncommon. Finkler has seen a man who suffered for months after influenza from hyperidrosis which nearly drove him frantic. When the man stood naked in a cool room the perspiration would literally pour in streams from his skin. It is interesting to note that in this case atropin had no effect whatever, while the attacks suddenly disappeared after salipyrin was administered.

The Kidneys.—Albuminuria is by no means uncommon in influenza, but nephritis must be looked upon as an infrequent complication or sequela. When it occurs it is a true acute infective glomerulo-nephritis—to use Klebs' term. Glycosuria has tolerably often been met with in or after influenza.

The Digestive System.—The digestive system suffers severely in many cases of influenza, but the disturbances are of functional or nervous origin rather than pathological in character. The thickly coated tongue, foul mouth, and fœtid breath have already been mentioned. There is sometimes absolute loss of appetite, amounting to disgust at the very idea of food, constipation, flatulence, vomiting, and less frequently diarrhœa. Occasionally tenderness on pressure and pain in the ileo-cæcal region indicate the presence of a true influenzal enteritis. If to these symptoms distension of the abdomen, insomnia, delirium, rapid pulse, and enlargement of the spleen be added, the diagnosis between true enteric fever of a severe type and the *enteric* or *typhoid* form of influenza becomes most difficult. Widal's test for enteric fever may help the physician to a correct opinion. Finkler points out that in these cases the anorexia and accompanying gastric symptoms may last for weeks after the influenza has declined, so that a notable loss of flesh and even a grave cachexia may take place.

The liver is rarely much affected in influenza. It may, however, be the seat of congestion or of a parenchymatous degeneration, such as occurs in other acute toxæmias. Marked jaundice is also rare, though an icteric tinge of the conjunctivæ is often seen. Dr Bäumler of Freiburg met with slight jaundice so frequently that he regarded it as a valuable diagnostic symptom of influenza. Still he admits that he could not demonstrate the presence of bile pigments in the urine, and the "jaundice" was confined to a yellow coloration of the sclera.

The Nervous System.—"There is hardly a nervous symptom in existence," says Finkler, which has not been observed in influenza. He proceeds to instance headache, backache, neuralgia, myalgia, hyperæsthesia and anæsthesia of the cutaneous nerves, vertigo, insomnia, delirium, syncope, debility, post-grippal neurasthenia, hysteria, and hystero-epilepsy, coma (so that the malady resembles the sleeping sickness or noma of the West Coast of Africa), chorea, singultus, yawning, tetanoid spasms, and local paralyses. For these various nervous phenomena Finkler believes that we must seek an explanation in the action of some soluble toxin. He further believes that they are all purely functional disturbances, basing the opinion on the facts (1) that the combination of nerve symptoms with atrophy was not sufficiently marked to justify a diagnosis of anatomical changes in the nerves themselves; (2) that recovery has frequently resulted in such cases with astonishing rapidity; and (3) that drugs such as phenazone, salipyrin, or quinine frequently caused a change for the better, or even a perfect cure so suddenly "that the diagnosis of a functional nervous disturbance was forced upon us *ex juvantibus.*"

It must not be supposed that organic lesions of the nervous system are unknown in connection with influenza. Far from it. In the first place, this is one of the diseases of which the toxin damages the nerves. Peripheral neuritis, shown by pain, paræsthesiæ, and paralysis, may be present locally or as a multiple neuritis (peripheral polyneuritis). Or "nuclear paresse," as Leichtenstern well terms them, may arise from lesions of the motor nuclei of the third and fourth cerebral ventricles caused by the toxins of influenza.

Apoplectiform seizures have occasionally happened. In 1890 Leichtenstern reported eight instances, to which he afterwards added four more. The cerebral paralysis of influenza may show itself as a monoplegia or as a hemiplegia, and may be complicated by delirium, convulsions, and coma. This group of symptoms is probably due to microbic embolism of the vessels of the pia mater, similar to what may be observed in cases of epidemic cerebro-spinal meningitis and of ulcerative or malignant endocarditis. Fürbringer has reported two cases of grave organic cerebral affections following influenza, in which large hæmorrhagic foci in the brain were found after death. Similar focal diseases of the brain have been recorded by Bristowe, who observed five cases in which the preceding influenza was looked upon as the etiological factor in the encephalitic process (Finkler). In 1897 Pfuhl reported three cases of what he believed to be localisations of influenza bacilli in the brain. As these bacilli are now known to be capable of exciting suppuration, they may be the cause of primary purulent changes in the brain; or secondary purulent foci may result by metastasis from suppuration in the ear, frontal sinus, antrum, nose, or tonsil.

Purulent meningitis may be brought about in precisely the same way in or after influenza. At the same time Krannhals of Riga has observed a number of cases which clinically resembled an undeveloped cerebro-spinal meningitis, but which at the autopsy showed only excessive hyperæmia of the pia mater. In every such instance there was pneumonic infiltration of the lower lobes of the lungs. In quoting this observation, Finkler says: "It is of extreme importance to remember that we may have these marked meningitic symptoms, such as great fever, apathy, sopor, stiffness of the neck, strabismus, inequality of the pupils, grinding of the teeth, hyperæsthesia, bradycardia, and intermittent respiration, without any lesions whatever of meningitis."

Organic affections of the spinal cord are, according to Finkler, rare in influenza. Symptoms referable to a disseminated myelitis have been observed by Benno Herzog. Revilliod reports the case of a young girl who, after a relapse, became totally paraplegic, with symptoms of spastic spinal paralysis. The typical symptom-complex of Brown-Séquard's paralysis (hemiparaplegia with hemianæsthesia of the opposite side) was observed by Determann in a man aged 25. Acute ascending paralysis (Landry's palsy) has been described by Féréol and Laveran; acute anterior poliomyelitis by Teissier, Henoch, and Drashe.

Apart altogether from the brain disturbances, whether functional or organic, which have just been detailed, are the mental affections of influenza. The disturbances of the mind, or pyschoses, which attend or follow this strange malady, are countless in number and in variety. They form one of the most distressing, because one of the most intractable, features of the disease. While it is true that serious mental disease, temporary or permanent, may supervene on any acute specific intoxication, such as typhus, small-pox, or enteric fever, yet influenza easily takes first place as a predisponent to psychosis. Nor is it only in those who are by heredity or by constitution predisposed to mental infirmity that influenza plays havoc with the mind. Dr. Julius Althaus examined the question of predisposition with great care, and found that there was a congenital or acquired neurotic tendency in 62·8 per cent of the cases of post-influenzal psychosis, but no such tendency in 37·2 per cent of such cases. Kirn divides influenzal pyschoses into febrile and post-febrile, according as they declare themselves during the fever or after it has run its course. Delirium, attended by motor excitement, hallucinations, and sensory illusions, is the form which the febrile variety usually takes. States of mental exaltation and of depression follow each other in quick succession. There is insomnia. Recovery usually occurs, but the patient's memory may be a blank so far as his illness is concerned. The post-febrile psychoses, according to Kirn, are—acute mania, coming on immediately after the fever; melancholia and depression, appearing within a few days to a few weeks after influenza; and general paralysis, coming on as late as six months after the attack.

In addition to these graver diseases of the mind, we must note the not infrequent occurrence of either hysteria or hypochondriasis in the later stages of influenza. The following are extracts from a letter written by a victim of influenza in January 1900. The patient was a gentleman, aged about twenty-seven, who subsequently regained his usual excellent health. He writes:—

"As I am quite satisfied that the complaint I have (and many others) is something quite new in this country, I thought you would not mind my writing to let you know how matters stand with me. . . . I had a fair night's sleep—one or two 'heats and colds.' The next day I was not so well, and on Wednesday morning I went to Greenore Hotel. I walked on the shore after lunch, and had a very light dinner—fish and chicken. Having had some champagne at 4.30 P.M. I drank nothing at dinner, but took about a wineglassful of whisky punch at 10 o'clock. I slept the night through without any apparent changes of temperature, and felt as well as ever I did in the morning, when like *a fool* I took a tepid bath. This, I think, gave me a chill again, as, though I felt well all the morning and walked on the shore, I felt very dull towards evening. During the next night but one I was very dull and cold—nothing seemed able to warm me, and I got a bad attack of 'shivers.' By the way, when at Greenore, a young friend of mine was *wheeled* into the coffee-room of the hotel with all power of his legs gone, and he has had just the same symptoms as myself. . . . I need hardly say what a shock this gave me. . . . Some of the doctors looking after this case I mention told him to go out as much as possible; others told him to stay in—anyhow, the poor chap can keep no food in his inside, and cold is no name for him, though now and then he says he feels *hot!* This fever seems to me more like what fellows tell me they get at places on the West Coast of Africa—such as Bonny, Cape Coast Castle, and Lagos—for as far as I am concerned I have no sign of 'a cold' whatever. . . . If I am any way better to-morrow (Saturday) I will go home, as I am a bother here at night when I can't keep warm, even with a roaring fire. I hope you will not think this letter a great bother to read, but, as I said, I think these cases are new and interesting. Young M. of —— is another case just the same—a strong, hardy chap, and run down to nothing without any cold, and nothing to bother him in the world. . . . The only thing I am taking now are 'Carrara pills,' to keep me clear. Some of the cases seem to be taken the other way. My appetite is excellent, and especially for breakfast, for which I take porridge, boiled eggs (2), milk, and about a quarter of a cup of tea."

This letter illustrates very well the intensely subjective or self-conscious mental state of the convalescent from influenza. In a case recently under observation, a strong young fellow of twenty-two stated that he had never been well since an attack of influenza five months previously. His heart thumped at night, and flushes of heat would pass from time to time up his legs and thighs, finally affecting his head and face, and causing his temporal arteries to throb. These vaso-motor phenomena are very common, and cause infinite distress of mind as well as body.

Pathology and Anatomical Appearances

There is little that is distinctive or characteristic in the morbid anatomy of influenza. The disease kills through its complications and sequelæ, not by its primary effect on the tissues and organs. Hence the pathological appearances belong in a great measure to the secondary affections. Pneumonic consolidations in the lungs, which are often lobular rather than lobar; lesions of the pleura, of the bronchial tubes, of the myocardium,—all these are met with in many cases. But in the merely partial development of a specific pathology influenza closely resembles typhus fever. The chief feature is a widespread hyperæmia and catarrhal swelling of the mucous membranes lining the respiratory passages from the Schneiderian membrane to the bronchioles. Similar changes, though less marked and of less frequent occurrence, are observed in connection with the stomach and intestines.

The mucous membranes of the larynx, trachea, and bronchi show a redness of varying intensity (Ribbert), and, as a rule, are smeared with a tenacious grey or yellowish-green mucus. While these are the macroscopic appearances, Ribbert describes a very marked cellular infiltration of the mucosa, together with an engorgement of the blood-vessels,—these microscopic appearances giving one the impression of an erysipelatous process. This impression is strengthened by the fact that these catarrhal changes in the mucous membranes show a strong tendency to advance—spreading from the nasal passages and pharynx to the larynx and trachea, and bronchi, or in the reverse direction outwards from the bronchi to the larynx, pharynx, and nose.

In the lungs, the consolidation presents the appearance of splenification rather than of hepatisation. The cut surface of the pneumonic areas is usually smooth, wanting the minutely granular or granite-like appearance of a typical fibrinous pneumonia. Pfeiffer, as a result of his microscopical investigations, is of opinion that each infiltrated area of influenzal pneumonia stands in direct relation to a diseased bronchus. The epithelium of such a bronchial tube is infested with influenzal bacilli, which also lie in masses under the epithelium. The same observer has also found the purulent exudation in influenzal pleuritis filled with great numbers of bacilli.

As regards the heart, thrombosis within its cavities, endocarditis, and pericarditis are occasionally found. Weichselbaum particularly states that in a number of cases the myocardium showed a distinct fatty degeneration.[1]

The spleen, liver, and kidneys are not often profoundly altered in influenza.

[1] Finkler, *Twentieth Century Practice of Medicine*, vol. xv. p. 106.

As regards the nervous system, Jürgensen describes intense hyperæmia of the membranes of the brain. A. Pfuhl of Cassel states that he found the bacilli of influenza within the blood-vessels of the brain and spinal cord in every one of a series of cases showing congestive and inflammatory changes in the cerebral and spinal meninges. Foà of Turin found in a case of influenza, fatal by broncho-pneumonia and pulmonary hepatisation, numerous microscopic hæmorrhagic foci throughout the spinal cord, and in some places even degenerative changes. The hæmorrhagic areas were situated especially in the posterior horns almost invariably at the periphery, the degenerated areas for the most part in the lateral horns. The grey substance and the anterior horns were not in the least altered. Hetweg speaks of an influenza hyperæmia which affects the entire central nervous system. He regards the process as not an ordinary vaso-motor disturbance, but a forerunner of an inflammation.

DIAGNOSIS

Influenza must be distinguished from dengue, acute lobar pneumonia or pneumonic fever, cerebro-spinal meningitis, measles, or other eruptive fever in childhood, and enteric fever.

Dengue fever is a tropical or subtropical disease. Its epidemics are circumscribed. The fever movement is much more decided than that of influenza. Complications seldom occur. An erythematous rash is of very constant occurrence. In contrast to dengue influenza is a non-eruptive fever. When rashes do appear they are accidental rather than essential or specific, and they result from hyperpyrexia, or profuse sweating, or from the ingestion of such drugs as quinine, or phenazone, or salicylate of sodium. In the epidemic of 1889-90, in addition to several cases of herpetic eruptions, the writer saw three cases of papular sweat rashes with sudamina, and one case with erythema fugax.

As to pneumonic fever, it is only in those cases of influenza which are complicated with pneumonia that any doubt arises. The diagnostics are—the strict lobar distribution of pneumonic fever, its entirely cyclic course, its viscid, rusty sputum, its critical defervescence. The features of influenza pneumonia have already been given in this article.

Professor A. Netter, of the Hôpital Trousseau, Paris, in his article on "Cerebro-spinal Meningitis" in the sixteenth volume of *Twentieth Century Practice*, points out that influenza in its nervous form presents certain symptoms which resemble very closely the symptoms of that disease. These are especially the pain in the head and the back, the general soreness, and not infrequently delirium and vomiting. The onset in both cases is usually equally sudden. Influenza may even be accompanied with suppurative lesions of the meninges as Eugen Fränkel observed in one case. To add to the difficulty epidemics of both cerebro-spinal meningitis and of influenza have coincided. Netter is persuaded that the two diseases have been confused a number of times, even in Paris, and that the cases of "grippal meningitis," upon the frequency of which several writers have remarked, were actually instances of epidemic cerebro-spinal meningitis. The diagnosis would turn on the presence of Kernig's sign of meningitis, and the result of a bacteriological examination of the cerebro-spinal fluid withdrawn by lumbar puncture, as advised by Quincke. Regard, lastly, should be had to the character of the prevailing epidemic.

Dawson Williams observes that influenza, if accompanied by much coryza, may resemble very closely the onset of measles, and the resemblance may be the closer if there be much depression and high temperature. The most characteristic single symptom of the prodromal stage of measles is photophobia. In a child who has not previously suffered from measles the sudden onset of coryza, accompanied by dread of light, would raise a very strong presumption of measles, and it would be imprudent to hazard a suggestion of influenza even if that disease were prevalent. When the erythema of the palate (exanthem) appears early, it will be of assistance in confirming a diagnosis of measles.

In the epidemic of influenza of 1892, very many cases were observed by Forchheimer of Cincinnati, in which a differential diagnosis between scarlatina, rubella or rötheln, and influenza proved difficult at the beginning. After the cases began to increase in number the differential diagnosis seemed easier.

The diagnosis between enteric fever and the gastro-intestinal type of influenza will be aided by the application of Widal's test, and by Ehrlich's diazo reaction in a less degree. Attention should be paid to the prevailing epidemic. The presence or absence of rose-spots should be noted. The condition of the spleen should also be ascertained. The duration of the fever is greater in enteric fever.

TREATMENT

The *treatment* of the affection we have been considering is based upon common-sense principles. It is expectant, palliative, symptomatic. There is no specific for influenza; nor up to the present are there means at our disposal for securing immunity against its infection. The susceptibility to the disease varies remarkably from time to time in the same individual. It has already been shown in this article that immunity is not acquired by an attack, rather the reverse.

The *prophylaxis* of the disease turns on strict isolation, however difficult to secure. In the next place, the mouth and nose should be systematically and frequently disinfected by antiseptic inhalations, sprays, and washes. From his own experience in practice, the writer can speak most highly of quinine and resorcin among remedies of the class named. A grain of sulphate of quinine mixed with (but not dissolved in) a wineglassful of cold water makes an excellent and efficient aseptic gargle. The antimicrobic properties of quinine are well known, and its topical use after the manner just described at once relieves the symptoms of sore throat, which result from the strain of the phagocytic conflict in the tonsils—our first line of defence against the invading pathogenic microbes. Another excellent preventive is the frequent employment of a resorcin spray. Eighty grains of resorcin, dissolved in half an ounce of glycerine and seven and a half ounces of peppermint water or rose-water—such is the formula which has proved most useful as a mouth spray and also as a nasal spray. Inhalations of oil of eucalyptus, thymol, oil of mountain pine (*Pumilio*), and the like are also valuable aids in prophylaxis. Internally quinine has been given with apparent success as a preventive by Graeser, of the Polyklinik of Bonn. Finkler quotes Graeser's observations as follows:—

"In Bonn all the men of one squadron (of a regiment of hussars stationed at that place) were given 0·5 gm. (7½ grains) of quinine in 15 gm. (half an ounce) of whisky for a period of twenty-two days. During this experiment there became ill in the first squadron, 22 men; in the second, 7; in the third, 19; in the fourth, 42; and in the fifth, 32. The second squadron was that in which the quinine and the whisky had been given to the men. Out of the seven cases of sickness occurring in this squadron, three may fairly be excluded, because the disease made its appearance after the termination of the experimental period. The four remaining cases occurred on the second, fourth, fifth, and sixth day respectively of experimentation; after this period no more cases of influenza occurred in the second squadron while the quinine was being given, although the disease continued to progress in the others. This result cannot be due to chance."

During an epidemic of influenza in the winter of 1899-1900 in Manchester, the sanitary authority of that city, at the instance of their medical officer of health, Dr. James Niven, D.P.H., issued a placard containing the following "Precautions against Influenza":—

1. The sick should be separated from the healthy. This is especially important in the case of first attacks in a household.

2. Discharges from the nose and mouth should not be allowed to get dry on a pocket-handkerchief, or inside the house or workshop. They should be at once collected in paper or clean rag and burned. If this cannot be done, the paper or rag containing the discharges should be dropped into a vessel containing water.

3. Infected articles and rooms should be cleansed and disinfected. (See "Disinfection," vol. iii.)

4. Those attacked should not, on any account, join assemblages of people for at least a period of ten days from the commencement of an attack, as they are likely to convey the disease to others. In severe cases the person attacked should remain away from work for a period of three weeks from the onset of the disease.

5. During the epidemic special attention to cleanliness and ventilation should be shown in factories and workshops. Workpeople are advised to wear warm clothing and to avoid unnecessary exposure.

6. Persons who are attacked by influenza should at once seek rest, warmth, and medical treatment; and they should bear in mind that the risk of a relapse with dangerous complications constitutes a chief danger of the disease.

7. The attention of employers is especially called to these recommendations.

Dawson Williams, in an excellent article on "Incubation and Infectiousness," in *Twentieth Century Practice* (vol. xiii. p. 383), holds that the period of isolation ought to be a week to ten days after the commencement of the disease, according to the severity of the attack. In cases complicated by pneumonia it should be extended to the end of convalescence.

When a person is struck by influenza, only one course lies open to the patient, and that is, to take to bed with the least possible delay. Rest, warmth, and quiet are three sovereign remedies for the primary disease and the best preventives of its more deadly complications. We have said that there is no specific for influenza. True, yet many drugs play a useful part in its palliative and symptomatic treatment. Tonics, like quinine and strychnine; antineuralgics, such as phenazone (antipyrin), phenacetin, acetanilide; heart medicines, like digitalis, strophanthus, caffein; antiseptics, such as phenol (carbolic acid), salicylic acid, salol, salipyrin, all these and other similar remedies fulfil the several indications for treatment which influenza in its varying phases presents.

The most popular form for the exhibition of quinine in this disease is the ammoniated tincture, which may be given in one-drachm doses, equivalent to a grain of quinine, every four, six, or eight hours. After the first two or three days the official solution of quinine hydrochloride in tincture of orange peel may be given instead in like doses. Children take a grain of quinine mixed with and partially dissolved in milk without demur, especially if a little syrup of orange or syrup of orange flower water is added to each wineglassful of milk. Quinine salicylate may be given in wafer cachets containing three or five grains as required, where the combined effects of salicylic acid and quinine are desired.

In the heart-palsy and tedious weakness of convalescence of influenza strychnine is our sheet-anchor. In failing heart it is best given hypodermically—one twenty-second of a grain (=5 minims of the official solution) night and morning, or every eight hours. In convalescence the following will be found an agreeable mode of administration:—℞ Liquoris strychninæ hydrochloridi ʒss., acidi hydrochloridi diluti ʒiss., tinct. aurantii ʒvj., aquæ chloroformi ℥vij. M. ft. mist. *Signa:* "One-eighth part twice or thrice a day with or after meals."

In this prescription dilute phosphoric acid may be substituted for the dilute hydrochloric acid, and tincture of quinine for the plain tincture of orange peel. Also, it is advisable to interrupt the taking of this mixture after a few days, should it cause headache or the spasmodic pains of strychnine-accumulation.

While hypodermic injections of strychnine, combined with morphia if the patient is restless or sleepless, tell best in the emergency of heart-palsy, digitalis, strophanthus, or caffein may be exhibited when the crisis of heart-failure is past. From two to five grains of caffein may be given every six hours in the more acute stage, a quarter of a grain of sulphate of spartein being added to each dose, if so desired. As caffein increases arterial tension, trinitrin, which so markedly reduces it, may (if necessary) be exhibited at the same time. But the use of the nitrites in cases of extreme weakness of the heart must not be indiscriminate or unwatched.

Camphor elixir, composed of spirit of camphor ten parts, syrup five, and distilled water one part, mixes well in water and proves an excellent diffusible stimulant, in doses varying from half a drachm to one drachm. An American writer, in the *Boston Medical and Surgical Journal*, recommends, in the crisis of the weak heart of pneumonia, fifteen minims each of tincture of valerian, spirit of lavender, and aromatic spirit of ammonia, every quarter of an hour, until the pulse shows that the heart is regaining tone and beating more efficiently. The external application of warmth over the præcordial region is often attended by the happiest results when the heart flags.

When choosing between digitalis and strophanthus it is well to remember that the latter acts more quickly than the former. It should not, however, be forgotten that a remarkable tolerance for digitalis is established in acute asthenic pneumonia; indeed, in some cases this drug seems to exercise an almost specific action on the disease.

Liquefied carbolic acid in 2-minim doses, three times a day, proved an advantage in the initial stages of influenza in 1890-91.

Salicylate of sodium, given in combination with effervescent citrate of caffein, is most useful as a mild aperient and in relieving the rheumatoid pains of influenza. Salicylic acid and phenol may be exhibited in the form of salol, or better still, salophen (Drewes of Hamburg). The dose of the latter compound is 10 to 30 grains daily, or even more. Another salicylic acid compound is salipyrin, which contains 57·7 per cent of that acid with 42·3 per cent of antipyrin (phenazone). This last named coal-tar derivative is an invaluable anti-neuralgic when given in moderate doses. A favourite prescription is the following:—℞ Phenazoni gr. 12-20, tincturæ gelsemii ♏xx., aquæ chloroformi ad ℥ij. M. ft. haustus. *Signa:* "One-fourth part every second or third hour until pain is quieted."

A long experience leads the writer to the conclusion that phenazone even in very small doses is not a suitable drug for young children, in whom it produces cyanosis and other signs of collapse. Phenacetin is a drug of the same class which is safer, and which soothes to sleep while producing more lasting analgesic effects. It is but sparingly soluble in water, an objection which applies also to acetanilide (the so-called "antifebrin"), though to a less extent. A favourite and efficacious method of administering acetanilide is in the form of "antikamnia" tablets, which are found to contain about 70 per cent of that drug together with about 10 per cent of caffein and 20 per cent of sodium bicarbonate.

If the tormenting and sometimes excruciating pains of influenza are once relieved by one or other of the foregoing remedies, temperature is likely to fall, and the patient may be soothed to sleep. But a solemn warning is needed against any undue interference with the fever of influenza or any other febrile disease. The nature of the fever process is now far better understood than it was even a few years ago, and we have learned that "fever," or elevation of bodily temperature above the standard of health or "normal," serves a useful purpose, provided that it is properly controlled. There is, in fact, what the Germans aptly call "das Heil-Fieber"—"the fever which brings back health." At the close of an able address on "Antipyresis" before the Tenth International Medical Congress at Berlin, in 1890, Professor Arnaldo Cantani of Naples used the memorable words—"Das Fieber, das in so vielen Krankheiten der beste Verbündete des Arztes ist"—"the fever, which in so many diseases is the best ally of the physician." Fever, in a word, purges the system. Therefore, high temperature should not be unduly meddled with, particularly in influenza, when it so quickly spends itself, as a rule.

The dieting of the influenza patient will often test the physician's skill and tact far more severely than the mere prescribing of medicine. Of this disease it is especially true that there are no two patients whom precisely the same dietary will suit. We might say: *Quot homines, tot epulæ.*

Of course the dietetic rules which apply to any febrile disease apply equally to influenza. Slops at first, solids later on in a gradually ascending scale, from lightly boiled fresh eggs to chicken, sweetbreads, lamb's fry, trotters, tripe and cow-heel, fish, roast joints, etc. But there are cases in which even the lightest foods are spurned with loathing. Then water—cold or hot—may be sipped, or "egg water" (the *eau albumineuse* of the French) may be given. This excellent "dish" is prepared by blending with a pint of cold water the whipped-up whites of from two to four eggs, and flavouring with salt or cinnamon. White wine whey may be given with equal parts of unflavoured egg-water, if a stimulant for a very delicate stomach is desired. A more substantial meal is egg-flip. The lightest form of this is made by pumping soda-water from a siphon on to the whipped-up yolk of an egg. Then the animal broths may be given, always remembering that an almost infinite variety of flavour may be obtained by mixing different broths together—for example, chicken-broth and beef-tea in equal parts. In a word, in attempting to draw up a bill of fare for a fastidious influenza patient,[1] we should always remember that "what is one man's food is another man's poison." Dr. T. King Chambers, in his excellent *Manual of Diet in Health and Disease* (published in 1875), reminds us that when the tailor in Laputa sternly refused to take the usual measurements, and insisted on constructing Captain Gulliver's coat, waistcoat, and breeches on abstract principles, the customer vowed it was the worst suit of clothes he ever had in his life. Dr. Chambers adds: "We should certainly fail in the same way if we did not take the measure of numberless contingencies in the daily life, and numberless peculiarities in the persons of those who consult us about their diet and regimen."

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[1] See "Invalid Feeding."

Always true, these words apply with special propriety to the dietetic treatment of influenza.

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Insanity.—The subject of Insanity is here discussed in the following four main sections:—

1. **Etiology.**
2. **Pathology.**
3. **The Nature and Symptoms.**
4. **Treatment.**

The following articles will be found in other portions of the work:—

- **Adolescent Insanity.**
- **Alcoholic Insanity.**
- **Choreic Insanity.**
- **Civil Incapacity.**
- **Climacteric Insanity.**
- **Criminal Responsibility.**
- **Dipsomania.**
- **Epileptic Insanity.**
- **General Paralysis.**
- **Homicide.**
- **Lunacy.**[1]
- **Masturbational Insanity.**
- **Mind.**
- **Paralytic Insanity.**
- **Paranoia.**
- **Pregnancy (including Lactation and Puerperium).**
- **Senile Insanity.**
- **Sexual Perversions.**
- **Sleep.**
- **Suicide.**
- **Unconsciousness.**
- **Uterine and Ovarian Insanity.**

Insanity, Etiology of.

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Heredity and Stress.—The causation of insanity is a highly complex question in detail, yet the broad fundamental facts may be narrowed to two comprehensive statements regarding heredity and stress. The inherent faulty nervous organisation of the individual and the stress of environment constitute the basis of our considerations in regard to this subject. How far these considerations may lead us even in a brief survey of practical import must be evident, as they relate to all the various forms of systematic knowledge which are subordinate to the master art of healing. In elucidating the etiology of insanity we have not only to specialise in the department of psychiatry, we have to pass beyond its limits, and regard the question from the point of view of general medicine, we have to investigate sociological conditions, and pass in review large questions of education, of training, of hygiene. In short, if the science and art of medicine is to be of value in elucidating the causation of insanity, it must reveal the past, predict the future, and so order that prevention shall minimise the cases presented for cure. Cure comes late in the day, we are confronted with an intolerable mass of confirmed mental disorders from which recovery is impossible. That well-worn adage *obsta principiis* is the watchword of the medical profession, and to the prevention of disease our energies must be directed. Is insanity, then, one of the preventable diseases? Can we stamp it out as typhus has been subdued by hygienic control? Not so long ago phthisis was permitted to ravage the country, yet to-day we have high hopes of altering its incidence altogether, we are even impatient that it has remained so long with us. True, there is no gross demonstrable bacillus at work in the ordinary case of insanity. We have to trace the history of failure beyond the individual, and to deal with a more complex causation; yet in a larger hope for future generations. Were it not so, our labours in research would be vain, our counsel untrustworthy and misleading.

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[1] Specially dealing with practical points in the notification.

Definitions, Legal and Medical.—Many definitions of insanity have been put forward, and these need not be discussed here. The all-important fact is, that the brain is the organ of mind, and that mental disorders are to be correlated with the nervous system, more especially with the frontal lobes of the cerebrum. We need not enter upon a discussion of the conceptions of mental action. At any rate it in some way coincides with intimate and subtle changes in the nervous system. Face to face with the practical administration of law certain definitions are requisite, and these have been modified from time to time as knowledge grows, until they have become propositions which fairly represent insanity in sociological relations. As to criminals who are insane, the law requires to be informed if they knew the nature and quality of the criminal act, and that it was wrong. That is being further developed by the question, "Could he help it?" If the crime under review were committed by a person who did not *know* what he was doing (and Sir James Fitzjames Stephen imports a metaphysical distinction into the word *know*), or if he could not help it, then he is regarded as legally insane. Or, if a person executes a will which can be shown to have been made under the influence of ideas repugnant to the common sense of society, he is held to have been of unsound mind, memory, or understanding. Lastly, the law may inquire whether a person is capable of managing himself or his affairs, and evidence is led to show what his conduct has been. Should his conduct be, in certain measure, different from what is commonly held to be right and prudent, it will be held insane, and he will be placed under judicial guardianship. The law takes but little cognisance of trifling deviations from conventional usage, and its definitions are designed to meet cases of patent insanity. Medicine, on the other hand, is less easily satisfied, and a medical definition of insanity becomes prolix in the endeavour to prove comprehensive. Small deviations are often momentous in consequences, but the superficial observer is impatient of the "trivialities" of science. Yet, as the twitching of a finger may be the herald of gross cerebral mischief, so may a trifling irritability grow into a mental storm of intensity and duration. The little cloud like a man's hand foretells the deluge.

As the world becomes more critical of *conduct*, so is the alienist most observant of *conduct*. The insane person is antisocial, or rather unsocial. He does not play his part as formerly, he does not play his part in conformity with the rules of the game. Dr. Mercier has summed up the whole matter in "three factors—disorder of the highest nerve arrangements, disorder of conduct, disorder of consciousness; and in every case the disorder of consciousness includes disorder of thought and feeling, of self-consciousness, and of consciousness of the relation of self to surroundings."

A consideration of Cullen's definition of insanity shows how far we have advanced. He said: "Insanity is a lesion of the intellectual faculties without pyrexia and without coma." Although we still speak of the intellectual faculties our business as physicians is with the underlying physical constitution; and Cullen's definition fails as completely in regard to the stated lesion as in regard to the absence of pyrexia. We are not now concerned with mere imperfections of the intellect, our measure of insanity is the observed reaction of the individual in relation to his environment; if that reaction is abnormal, owing to nervous disorder, the person is insane, and it is this question of disorder consequent on disease which makes insanity an affair of medicine. The disease may be of the most varied character. It may be what we conveniently term functional, it may be a coarse degenerative process, it may be an arrest of development. And in consequence of these infinite variations, from the indefinite beginnings of abnormal action to the most marked depravation of mind, there are innumerable cases of difficulty, elusive and changeable. Thus we have to fall back upon the rule to consider each case on its merits—the circumstances affecting each have to be weighed. Further, for our purpose in elucidating the causes of insanity we have to take into consideration the neuroses which are indicative of nervous instability. We cannot in this connection exclude these "minor" disorders, such as neurasthenia or hysteria. The neurotic inheritance by transmutation may evolve as marked insanity. The seeds of degeneracy are evident, and are only too apt to germinate.

We have arrived, therefore, at the conclusion that nervous instability, nervous derangement, nervous degeneration, are the underlying physical facts of insanity; that the mental aberration is at least concomitant with these facts, and that we have to deal with the various forms of insanity on the same general principles which guide us in other departments of medicine, specialising, indeed, as may be requisite in the due observation of nervous phenomena, and giving all attention to the details of conduct which constitute the reaction of the individual whose mental condition is the subject of investigation.

Pathological Considerations.—The anatomical, physiological, and pathological considerations which call for special study need not be enlarged upon here (see p. 306). Suffice it to say that these are of the utmost importance, and that the present state of knowledge is advancing rapidly year by year. We may, however, but briefly indicate that the nervous system is liable to disease in conditions similar to those occurring in other parts of the body. If there be a difference it is in the far-reaching effects of toxic agencies, traumatic accidents, inflammatory and degenerative processes affecting the delicate and complex constitution of the most highly organised and latest developed material in nature. The neurons with their complicated connections are susceptible of damage in measure yet undetermined by our finest instruments of research. Grosser changes, indeed, have been detected where, but lately, changes were denied. The territory of functional and idiopathic disorders is being rapidly investigated, and pioneers are charting the unexplored. We are thus enabled to comprehend the fatuity of advanced epileptic dementia, and the hopelessness of recovery, long recognised, is explained by the irreparable damage to the nervous elements. We are thus impressed by the primary importance of physical conditions which eventually become manifest in mental disorders. The mental disorder, the insanity, is not the main question in the mind of the physician. The grandiose speech and the extravagant conduct of the general paralytic are merely symptoms of a special cerebral disease. They do not constitute the real malady, however helpful in diagnosis. The general paralytic may be depressed and melancholic, but he is still suffering from general paralysis. The essential nature of his trouble is the cortical disease of his brain. And so it is with the whole group of nervous diseases, we may be struck by the grotesque gestures of chorea, we may be appalled by the convulsions of epilepsy, we may be observant of the attitude of stupor, but we must pass beyond these apparent phenomena to attain a true understanding of the fundamental and real disorder. We must not be over-much occupied by the consideration of the mental symptoms of insanity, we must search for their causes in the ancestors of our patients and in the life-history of the patients themselves.

Causes stated.—The Scottish Commissioners in Lunacy have hitherto refrained from reporting upon the causation of insanity on the ground that the information obtainable is altogether untrustworthy. The English Commissioners, however, compile tables in regard to causation from information verified by the medical officers of the asylums. These tables show the percentage to the yearly average number of patients in the private and pauper classes admitted. During the five years 1892-1896 the table stands thus:—

	Private.		Pauper.	
	M.	F.	M.	F.
Domestic trouble	4·5	10·7	4·2	8·8
Adverse circumstances	8·3	3·9	7·2	4·5
Anxiety and worry	14·0	11·0	4·9	5·0
Religious excitement	1·7	3·1	1·4	1·5
Love affairs	1·2	3·6	·6	2·0
Fright and shock	·7	2·0	·8	1·7
Intemperance in drink	20·5	8·6	21·7	8·9
Sexual	2·3	·4	·9	·6
Venereal disease	3·9	·3	1·6	·5
Self-abuse	4·0	·9	1·8	·1
Over-exertion	·6	·6	·3	·2
Sunstroke	3·5	·2	1·5	·1
Accident or injury	2·9	·7	4·9	·9
Pregnancy	...	·7	...	1·2
Puerperal	...	6·5	...	6·4
Lactation	...	·8	...	1·6
Uterine and ovarian	...	2·4	...	·8
Puberty	·5	·9	1·0	1·0
Change of life	...	7·4	...	4·5
Fevers	2·4	1·8	1·3	·8
Privation	·2	·5	1·5	1·5
Old age	3·6	3·8	6·5	7·3
Bodily diseases	9·0	10·9	14·5	13·0
Previous attacks	15·4	23·5	16·7	22·0
Hereditary	20·9	29·0	20·7	25·7
Congenital	5·2	2·0	5·1	3·9
Other causes	1·7	1·2	1·0	·7
Unknown	13·3	9·3	18·4	16·1

These "causes" include varieties of stress consequent upon which the patients under cognisance of the Commissioners succumbed to mental diseases, as well as the enumeration of cases of an hereditary nature. It will be seen that the mental strain imposed upon the individuals told more heavily upon those of the private class than upon those of the pauper class. The domestic troubles, as might be expected, fall heavily on women, while men are more oppressed by business anxieties and worries. But these single "causes" by no means represent the whole truth. Trouble and worry are involved with many other disadvantageous circumstances, and issue in a deteriorated state of health which leads by easy transition to mental disorder. Turning to the physical causes, the preponderating influence of alcoholic intemperance is at once recognised, but one may doubt if the figures represent a close approximation to the truth. A more rigorous investigation materially reduces the percentage, but when the social effects of alcoholism are considered, we may place against that reduction the cases of domestic trouble and adverse circumstances consequent on the abuse of alcohol. On an examination of 48 alcoholic cases out of a total admission number of 520, the proportion was found to be 9·2 per cent at the Perth Royal Asylum, which deals only with private patients. These divided into classes as follows:—A. Alcoholic persons without other ascertained cause. B. Alcoholics who were hereditarily insane. C. Alcoholics with alcoholic heredity. D. Alcoholics consequent on physical stress. E. Alcoholics consequent on mental stress.

A	3·26	per cent.
B	3·07	„
C	2·11	„
D	0·58	„
E	0·19	„
	9·21	„

Or to put a concrete case. A gentleman, whose mother had laboured under recurrent mania all her adult life, informed me that he had for years averted periodical attacks of mental depression by a free use of alcohol. On his admission his symptoms pointed to alcoholic intemperance, but the ordinary motor symptoms soon passed off, leaving him in a state of acute melancholia. Alcohol had at last failed him. His alcoholism was the result of insanity, and by no means the cause of it. There are other facts, however, which demonstrate the widespread influence of alcoholic intemperance in the production of insanity, *e.g.* the well-known observation of Dr. Yellowlees who showed how good times and high wages filled the Glamorgan County Asylum as a direct consequence of excessive drinking, while the converse also held good.

By this table of the Commissioners the effects of bodily diseases, toxic states, crises of life, old age, are also shown approximately. It will be seen that many had previous attacks of insanity in the proportion of 15 or 16 in men to 22 or 23 per cent in women. Lastly, we may remark the percentages stated in regard to heredity, which are assuredly understated owing to the difficulty of obtaining information as to the intimate family history of the cases coming under observation. At any rate, of patients received into the Perth Royal Asylum during the past eight years, the average proportion of those who had an hereditary tendency to insanity is 43 per cent, and to neuroses 19 per cent in addition. And yet this is not all the truth, for in spite of every care and repeated investigations there are patients of such a nature that their hereditary taint can be recognised in the symptoms of their insanity, but who are excluded from the list of predisposed either by the wilful untruthfulness of their relatives or by the absence of definite information.

Heredity and Neuroses.—We shall now consider in some detail how this chief cause of insanity occurs. The outstanding laws of heredity are well known, and have been the subject of much discussion of late years. Darwin and Weissmann have each their disciples, and with regard to the main fact they are agreed that there is a transmission of parental attributes. It is sufficient for our purpose to accept the laws of heredity in the Darwinian form, for we cannot but believe that slow and gradual modifications by environment are transmissible (see "Heredity," vol. iv.). It is nothing to the purpose to search for the transmission of imperfect limbs consequent on parental amputations. The law of solidarity (the transmission of ancestral characters intact) repels any such objection, which is merely a revival of early objections to the Darwinian theory of evolution. But, although the tendency is for the child to inherit every character of the parents, there are various modifying influences at work, such as prepotency. There are also toxic influences, such as syphilis and alcoholism, which are undoubtedly transmissible and alter the course of the life of the offspring. There can be no doubt that the child of parents advanced in life is endued with less vitality than the normal, and that pathological heredity follows the same line as physiological heredity. We recognise that it is not so much a transmission of pathological characters as a predisposition to succumb to pathological influences. The child is not born phthisical, but its constitution develops into a condition favourable for the growth of the special bacillus. Then, again, there are well-known laws relating to consanguinity and exsanguinity, which must be observed if heredity is to be maintained in the most favourable condition as regards offspring. This becomes a question of practical importance in the consulting-room, for it is occasionally asked whether a person with an hereditary predisposition to insanity should marry. The marriage of first cousins is not uncommonly proposed in families of insane heredity, for these individuals have an undoubted tendency to form such connections. These marriages should be forbidden if a common and near ancestor has been insane, or if there is a near inheritance in each of the separate families; but it is permissible if insanity has declared itself in only one of the separate families. The question of inbreeding has not been discussed with moderation at all times; no doubt if continued for generations it is disastrous; but Huth has shown that arguments and facts are wanting to prove that consanguineous marriages in healthy families cause insanity and similar evils.

Insanity is a retrogression, a dissolution. It is a reversal of evolution, and as such has its beginnings in the latest evolved structures and functions of the brain. The recently acquired characters are least stable; they have been added in course of generations by the operation of natural selection and the conditions of environment. It has been said that if men gave the same attention to the propagation of their species as they do to the breeding of horses the race would have been more highly civilised, more highly developed physically and mentally. But while that is in measure true, we must recognise that the successes of the horse-breeder are few compared with his disappointments. We are in haste to be perfect, but the operations of the laws of heredity are not abrogated on that account. And the fact remains that marriages are not entered upon with that sense of responsibility to offspring which is desirable.

Degeneracy.—What is degeneracy? Much has been written round about this subject, and we have had elaborate descriptions of the stigmata of degeneration which are held in light esteem by those who are definitely opposed to the extreme teaching of Lombroso and the pamphleteering of Nordau. Following Morel, and making a special study of the prisoners under his care in Perth, Bruce Thomson came to the conclusion that heredity was the prime factor of criminality, and that environment determined the almost inevitable issue. He outlined the physical appearances of criminals, called attention to their malformations of body and perversions of mind. These are the stigmata of degeneration familiar to those who are brought into contact with the criminal and the insane. They are pathological deviations from the normal type, not to be declared morbid separately. The weight of evidence declares them to be morbid, just as in the Tichborne case Orton was not found to be an impostor on one count or another, but consequent on the summation of all the counts, by the strength of the condemnation of all the evidence. It would indeed be surprising if mind did not betray weakness when manifested in a body stunted, malformed, and weakly in organisation. The nervous system is imperfect in development, and mentalisation is degraded in proportion. One would not expect a perfect mind from an organism thus morbid in constitution. The reactions of the criminal mind are morbid in proportion as the criminal is degenerate. We do not desire to push this conclusion too far, holding that it is but a proportion of criminals who are degenerate; but when that proportion can be shown to exist in prisons and in asylums we must recognise the facts, and endeavour to assess the amount of degeneracy when called upon in courts of law. For instance, in the case of Lawrie who committed a murder in Arran insanity could not be proved; but he was declared to be so far degenerate or abnormal that he should not be hanged; yet he was sufficiently master of himself to undergo the modified punishment which was awarded him.

This morbid heredity, however it has arisen, is transmissible from parents to offspring. It may not be a certain form of insanity—it is rather a certain morbid conditioning of the nervous system which may declare itself as a neurosis in one generation and as a typical insanity in the next. The neurotic alcoholic is likely to propagate epilepsy, and the children of a hypochondriac may suffer from suicidal melancholia. Indeed, there is often a similarity in the very form of the insanity transmitted, even in the mode of suicide in successive generations.

It will be seen, then, that heredity may be altered by careful or by fortunate selection, the morbid constitution may be improved by the prepotency of healthy new stock.

The causes of degeneration manifested in neuroses and insanity are not far to seek. Briefly, we may group them as faults of inheritance, of civilisation, of alimentation, and of toxic origin.

We have seen how the laws of heredity are illustrated in the production of insanity. In conformity with the general observation, we remark that exhausting diseases in parents are followed by a weak offspring, and in degenerates there is a failure to develop shown by harelip or other monstrosities.

The instability is the heredity, and the faulty nervous system is the first cause. The more we study the intimate family history of each insane patient the more convinced we are of these antecedent circumstances. In the incidence of influenza now so commonly assigned as a cause of insanity, there is hardly a case to be found where hereditary defect cannot be traced. Where, then, did this evil begin? Is it a mere variation transmitted by direct inheritance? We believe not. In the mobile and varying circumstances of human life, in the progress of the race through the centuries, there must be a certain proportion of human beings who fall below the normal standard. By the mating of these, their generations successively become more unfit for the struggle for existence. In sub-human groups they would disappear at an early stage, but we are so accustomed to the methods of civilisation that we are apt to forget this. In the degenerative tendencies of mental disease we see families extinguished, the last survivor dying in the asylum. It is the conservation of civilisation which, so far, preserves those who are unfit, and we shall have yet to consider whether civilisation has its remedy.

Civilisation no doubt has a share in the production of insanity, but it is far from certain if that share can be estimated. The negroes of the United States of America were remarkably free from insanity under more primitive conditions. That immunity has disappeared, and they are now to be found crowding the asylums of the Southern States. But there are definite agencies at work in this unfortunate state of matters. Tubercle and syphilis have found a nidus in the negro, and he is suffering degeneration on the physical side in proportion to the mental degradation. Here we have, within a few years, an object lesson as to the effects of heredity on Darwinian principles. The germ cells have been affected, and in spite of Weissmann's declaration that they are apart from the soma, the results of a vicious civilisation are apparent. For civilisation has its vices and defects, and our boasted attainments and superiority over the "dark ages" are in some respects expressions in an optimistic vein. In its train civilisation has brought crowded cities, the factory system, squalid homes, and an ill-regulated population; or, at the other extreme, an unhealthy plutocracy, with a vacant, idle, and irresponsible life, craving for daily excitements, and gambling health away in the hot pursuit of pleasure. The benefits of civilisation cannot bribe us to shut our eyes to these unseemly facts, but should rather induce us to avoid the perils of our time, and to improve the conditions of the race. It is unlikely that ancient civilisations held a better place in respect of human life and happiness, or that the causes now operative in the production of insanity were formerly unknown. What concerns us more is the question as to whether insanity is really increasing in western civilisation, and specially if the increasing burden is as alarming and unpromising as it appears at first sight to be. In some forty years the number of insane persons coming under the cognisance of the Lunacy Departments of England, Scotland, and Ireland approximately expanded by 70,000, 9,400, and 6,200 respectively. These are figures which seize the popular imagination, and raise questions as to the possibility of stemming the rapidly rising tide. We cannot but believe that there is some increase of the occurring insanity as well as a vast increase in the registered insanity. Dr. Ireland has shown that the number of deaths from diseases of the nervous system in Scotland has grown by about one-third since 1855. No doubt much of this increase may be explained away by a better system of registration and wider knowledge, just as it may be argued that the increase in insanity and its allied conditions is indicative of an evolutionary advance with which weaklings cannot keep pace. The question has lately engaged the attention of the State departments in lunacy, and their general conclusion is that there is no alarming increase. They have not arrived at this conclusion without painstaking inquiry, which showed that there were many points to be considered besides the simple fact of so many more insane persons to be reported upon.

Of course the vast proportion of these cases is classed as *paupers*, but it must be remembered that this is a most misleading statement. Mr. T. W. L. Spence has shown that only one-fifth of those so classed in Scotland really belong to the pauper class. It is in the nature of insanity to degrade not only in mentalisation, but also in social condition. Still it is true that where there are most paupers there are most insane, and it has been shown that if the larger number of cases come from the manufacturing towns, that number is balanced by the accumulation in the agricultural districts, where there is more stagnation, where the insanity is less curable, whence have been drained the more active spirits. The destruction of the poor is their poverty; the recklessness, the thriftlessness, the drunkenness of the lower classes involves instability of nerve tissue. The evil is apparent, and the remedy must come by an adequate appreciation of the laws of health and a different mode of life for the people.

Yet insanity is not entirely an affair of poverty and low life. The upper and middle classes have their burden, but in the general improvement in moral tone and the fuller appreciation of the benefits of mental and physical hygiene the causes are somewhat different. The direct cause of first importance in this respect is the over-strain to which men are exposed in the whirlpool of modern life. We may accept Dr. Farr's calculation, that the proportion between the upper and the middle classes on the one hand and the lower classes on the other is as 15 to 85. Certain it is that those occupations which are inseparable from mental stress have acquired a common notoriety in the production of disorders of the nervous system, and when to these is added a life of excess and dissipation, the result is a vast increase in cases of general paralysis, which is a disease of great cities and of great excess.

Turning to the next main cause, which is almost inseparable from other considerations, we may make but brief mention of malnutrition, starvation, and privations generally. It is a truism that the brain requires healthy blood in abundance. Whatever interferes with its due nutrition must issue in disorders of the nervous tissue more or less severe.

Toxic action of various kinds claims a more detailed notice. We may venture to divide these toxic agencies as follows:—

1. Exotoxic—alcoholic, plumbic, anæsthetic, cocaine, morphia, pellagra, ergot.
2. Autotoxic—rheumatic, choreic, gouty, diabetic, myxœdematous and cretinoid, uræmic, etc.
3. Microbic (?)—syphilitic, phthisical, influenzal, malarial, septicæmic.

It is manifest that these various degenerative causes would require a much more extended notice than can be given here, and we must refer to other articles in order to clear up the many points of interest. Broadly, in these various agencies we have something which is capable of inducing the degenerative conditions of the nervous system, to which we have already directed attention. Take exotoxic forms, such as alcohol and lead, the results of chronic alcoholic intoxication in the destruction of the chlorophil elements of the nerve-cells is matter of common knowledge. Lead has been demonstrated in the brain tissues consequent upon plumbic poisoning. The poison is virulent enough to destroy, and the enfeebled parents produce an enfeebled offspring.

Again, the autotoxic results of rheumatism, gout, etc., setting free malignant substances in the system by excess, or by derangement of metabolism, are familiar to all. Or, when necessary substances are deficient, as in myxœdema, the bodily welfare is threatened by deprivation. Insanity is thus caused by a defect as well as by an excess, and some of the most remarkable cures of cases of myxœdema have been recorded in asylums, where to the ordinary physical degeneration and infiltration a marked hebetude of mind, with delusions more or less pronounced, have been observed. The administration of thyroid substance rescued these sufferers by replacing in their systemic circulation that which had been wanting.

We have ventured to class syphilis, phthisis, influenza, and septicæmia as microbic agencies in the production of degeneration. In Russia syphilis is generally held to be a practically constant factor in the production of general paralysis, and there is in this country a growing tendency towards a similar conclusion. In every relation of physical condition syphilis is a powerful agent in degeneration of tissues, and we cannot but assign to it a very prominent position in this list. The other agents named are appropriately dealt with under their respective headings.

We thus arrive at the conclusion that there are very various and powerful physical influences at work in the production of degeneration, consequent on which we have manifestations of the various neuroses, the insane diathesis, the various forms of mental derangement, and the stunted development of idiocy in its various degrees.

Direct Causes.—Turning now to the more direct causes of insanity, which have been already referred to, we find that they are divisible into two main classes—those commonly spoken of as moral (mental) and physical.

The mental causes are the various kinds of mental stress to which men and women are ordinarily subjected. In practice it has been found that cases of insanity are referable to these classes—adverse circumstances, anxiety, worry, domestic trouble, fright and nervous shock, love affairs (including seduction), over-pressure and over-strain, and religious excitement. After what has been said little remains to be added in elucidation of these headings. It is not so much steady, hard work with intervals of relaxation that is the cause, as the worry and scurry of life, the anxiety as to means of livelihood, the real troubles of existence. The excitement of politics leaves little trace on asylum records, while a financial crisis surely sends its victims to asylum care; and although religious excitement does not figure largely in the statistics of insanity, yet many cases occur showing delusions tinged by the religious movements of the time.

Passing to physical causes—what has been said elsewhere of alcoholic excess need not be repeated. Excess of sexual nature, over-exertion, malnutrition, and toxic agencies are all regarded as fertile exciting causes. The physical stress is apparent in such cases. We also find cardiac lesions in a great proportion of cases admitted to asylum, directly correlating rheumatic degenerative lesions with nervous deterioration. Patients with cardiac incompetence also occur through over-exertion in athletics, affecting the nutrition of the brain directly. The lesions of epilepsy, general paralysis, organic diseases of the brain, are referred to elsewhere, and their connection with this subject is evident.

We have still to consider the results of peripheral nervous irritation in the production of insanity. Schroeder van der Kolk directed special attention to what he called sympathetic insanity. He found that a great number of his patients suffered from constipation and were relieved by suitable treatment. He therefore laid the blame of their mental troubles on the peccant great intestine. That view was of importance in directing attention to the physical condition generally, but it does not seem that it can now command any considerable amount of support. The constipation is rather attributable to the defective action of the nervous system, and is a mere symptom of a more widespread and basic disorder. Similarly, recent attempts to correlate the insanity of women with uterine and ovarian disease can meet with no marked approval. The modern and probably correct belief is, that the insane manifestations are due to disorders of the brain, and while it is very likely that certain of these disorders are due to toxic influences—perhaps owing to intestinal fermentation, or to deficient action of the ovaries—it would be absurd to confine our attention to the treatment of symptoms of peripheral irritations. We would not be misunderstood; the physical condition of every insane person demands the most searching investigation, adequate treatment of every departure from healthy action is imperatively required. While that is so, however, our later knowledge of nervous processes forbids us to lay stress on the peripheral conditions to the exclusion of the central mischief. And, further, these visceral neuroses must be studied in less complicated conditions if we would be successful in dealing with them in the insane. Professor Clifford Allbutt's lectures on this subject should be the book of reference in clearing the mind of specialism, and entering the freer air of general medicine.

There are also such causes as traumatism and sunstroke. These are of sufficient importance as to demand separate consideration. Dr. van Gieson has demonstrated by microscopical methods a very similar morbid process in the brain cells in both of these conditions. The shock is sudden and nutrition is immediately and gravely affected. We cannot, however, but entertain doubts as to statistics of the incidence of sunstroke, for a case who has been resident in tropical climates is never sent to an asylum without sunstroke being alleged as the cause. We would venture to suggest that malaria is far more efficient and common in this relation than sunstroke.

Again, in the evolution of the individual critical periods of life occur, and the stress of these periods tells heavily upon those who are burdened with a nervous inheritance. Puberty, adolescence, the climacteric, have each their special dangers, and it is in accordance with the laws of heredity that those most seriously affected by degenerative conditions will be earliest affected by insanity. Senility, on the other hand, if not premature, is the natural expression of failure of the brain after the exhaustion of a long life.

Yet again we observe a critical and exhaustive, if not a toxic condition supervening upon pregnancy, parturition, and lactation. These varieties of stress are familiar to every medical practitioner, and there is undoubtedly a disposition to minimise or overlook neurotic manifestations in connection with reproduction, a disposition which is a danger to doctor and patient. The physician should be urgent to guide matters aright when these abnormal signs first become apparent.

There yet remain two rather rare but interesting causes of insanity, viz. communicated and epidemic. Much has been written in reference to these cases, which have acquired a new and increased interest in connection with the later developments of our knowledge of hypnotic states.

The other considerations relative to the incidence of insanity might rather be held to fall under a statistical inquiry. The heaviest rate of occurring insanity falls on the years 35 to 45 in this country, but in the United States of America the greatest liability appears to be from 20 to 30. The question of sex is complicated by various considerations—the tendency being towards a slight increase of female cases, yet leaving the proportions nearly evenly balanced. With regard to condition as to marriage it may be concluded that, at marriageable ages, and in proportion to the population, considerably more married than single and widowed persons are admitted to asylums.

Prodromes.—This article would fail in practical value if some account of prodromes and prophylaxis were omitted. We shall therefore proceed to discuss the results of causation in their general and earlier manifestations, and give some indications of the measures which may be adopted with hope of success.

The penalties paid for inattention to the laws of health are heavy and various. They are not only exacted from the transgressor, but also from his progeny. The prodromata are of special interest, and demand early recognition and prompt treatment. Dr. Clouston has devoted his attention to the neuroses of development, and has set forth his observations in the Morrison Lectures for 1890, and separate articles will be found in this *Encyclopædia* dealing with epochal insanities. These may be briefly referred to as adolescent, climacteric, and senile. But the insanities of childhood and the neuroses of early life also demand attention, and preventive medicine must be called into action.

These symptoms are forebodings of nervous instability likely to eventuate in serious mischief, *e.g.* sleeplessness, headache, inability to perform mental work, loss of attention and self-control, unusual irritability, crazy reckless conduct, morbid fears and indecision. The nervous child is often precocious, a victim to night terrors, subject to convulsions consequent on derangements of digestion or the peripheral irritation of intestinal worms, or the troubles of dentition. The caprices, explosive rages (fulminating psychoses), and similar incidents, are often made light of by parents who, being themselves neurotic and unstable, are singularly unfitted to manage their own children. The morbid arrangement of the nervous fabric is forced upon our attention, the motor manifestations are evident, and conduct becomes so abnormal that it is hardly to be overlooked by the most superficial observer. These are assuredly indications for treatment, mental and physical. Separation from injudicious influences, the educational provision of a competent nurse of equable mind and adequate training, association with other children of normal development, are imperative. Here is a wide sphere for the employment of women, in educating and training children of self-centred, ill-regulated habits before these habits become fixed and unalterable. And the association of these children with others, so as to develop normal feelings with normal social instincts, is hardly less important. We must ever remember that these children are not merely nervous, but that in their hereditary misfortunes such facts as tuberculosis, gout, cancer, and other maladies may have a place. It is only of late that the cardiac diseases of childhood have been sufficiently recognised even amongst the leaders of the medical profession, and every asylum physician is familiar with the fact that cardiac complications are to be found in a very large proportion of the cases he receives for treatment. This wise guidance is a duty laid upon general practitioners; the specialist is called in too late when a definite malady is declared. We must look to the family doctor to direct the parents in their duty towards their children. That opens larger responsibilities than the apothecary dreamt of, but it is in strict accordance with modern tendencies, and it is thus that "doctors will become," in Gladstone's memorable saying, "the future leaders of nations." Year by year we must see the commonwealth advance in relation to the hygienic and moral control of individuals, not by sumptuary laws or impulsive legislation, but by a true education which will lift the people out of squalor and ignorance and fit citizens for the work of the nation. Thus education will no longer be anchored to ancient formulæ and circumscribed by dead ideals; the process of cramming will be thoroughly and finally discredited; and the individualising of each scholar will be the aim of enlightened teachers.

For the neurotic precocious child ordinary present-day education is a real danger. This has been partially recognised by the establishment of schools for backward children in Leicester, London, and elsewhere (see Mental Deficiency); but we have generally failed to control the forward child, whose lively memory and tricks of imitation carry it into the perils of a one-sided development of the nervous system,[1] and often result in disaster. The nervous system is all-pervading, and for the due exercise of every part education must be extended in order to develop every function. Seeing and hearing are necessary to a real assimilation of instruction; yet not only the printed type and the teacher's monitions, but all the avenues of knowledge must be opened up to permit of a general cerebral assimilation. As every muscle is represented in the cerebral cortex, so should every muscle be scientifically exercised. In short, the aim and object of all education must be to secure the sane mind in the sound body.

Hygienic.—That leads, of course, to the consideration of the dangers of unhygienic practices in forms familiar to all. At puberty, when there is danger of the degenerate, endued at conception with a deficient vital force, failing to develop, the precautions above indicated must be redoubled. There must be rest as well as exercise. The brain must not be allowed to rust in idleness, yet mental work must be moderate in intensity and in quantity, and physical excess must be equally regulated. In girls especially at this critical period great caution is required. Fatiguing muscular exertions should be absolutely forbidden, extremes of heat and cold should be avoided. The touchstone of success will be the establishment of physical health, the avoidance of anæmia, the broadening of interests, and the healthy avoidance of evil habits.

In regard to those who have to gain a livelihood comes the question of the choice of a career. That must be considered in view of the mental and physical aptitudes. It seems hardly necessary to advise that neurotic persons should keep as close to mother earth as possible, yet there is a decided inclination among such persons to decide for emotional, exciting, irregular modes of life; and it is difficult to induce them to be content with the even routine of a government office or the bucolic delights of the country, while the stock exchange or the crowded halls of commerce beckon them on with high promises that so often end in disappointment and disaster. "The land for the people" is a cry that comes with convincing appeal in these days of overgrown cities; and when one considers the stunted degenerates of town life, one cannot but re-echo that cry in the interests of the race. The career for neurotic individuals should be free from great responsibilities, should interpose no great difficulties, while it should afford occasion for the exercise of healthy volition. It should be restful in the intervals of work, yet it must be remembered that idleness is not rest—a change of occupation and a variety of interests are both necessary. Kraepelin has shown that mental work added to physical work is provocative of exhaustion; recuperative rest in intervals of brain excitation is not to be gained by bodily exercise. The nervous system is stimulated and exhausted by both these processes, and we have to fall back upon nature's sweet restorer sleep. We need hardly urge the importance of sound refreshing sleep, or indicate that it must be gained by natural means and not by toxic substances.

That brings us to a brief summary of conclusions as regards the use of alcohol in nervous cases. We have shown that it is a widespread cause of degeneracy, and that it plays a leading part in the production of insanity. We therefore prescribe it with grave unwillingness for the cases under review. Safety for a neurotic individual is only to be maintained by a total avoidance of alcoholic liquors or other drugs of lethe. It may be advisable in rare cases to give it as an anæsthetic in conditions of fatigue and in peril of collapse. It may be required in anæmic complications, but the possibilities of abuse render it a two-edged weapon which we shall do well to avoid using in so far as possible.

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[1] See article "Hysteria" in children.

There can be no doubt that those of feeble nervous constitution are liable to take extreme views in connection with religion, and guidance is required in this sphere of thought. The green sickness of youth is often characterised by bizarre opinions in religion and politics, which a greater experience of life modifies. Heinroth's theory of the common origin of insanity and sin was fortunately soon put aside by his fellow-countrymen; but every now and then it crops up amongst those afflicted with certain forms of religiosity. The religion of the present day is more concerned with conduct than with forms of profession, and the religion for neurotics is of a moderate, practical nature—avoiding undue introspection or the attempted solution of insoluble questions.

If a person of unstable nervous tendencies decides to marry, the most reasonable advice usually comes too late and is often taken amiss. For the women there are dangers in the probabilities of adverse circumstances and domestic troubles; in too rapid procreation; in the petty worries of life which marriage so often entails. For the men, there are dangers in the greater responsibilities and the social difficulties which they undertake to meet. But objections raised in the consulting-room very rarely carry weight, and until the state interferes to limit the procreation of the unfit, we can only rely upon right education informing the public mind and emphasising the moral code. At present, in this transition stage, it is evident that prudence in marriage is confined to the classes who have least need to exercise it. The criminal, the reckless, the improvident put no bounds to the gratification of their desires, and the breeding of paupers and inefficients is assuredly a danger to the commonweal.

Restrictive measures were not unknown in former days. Howell (*Familiar Letters*, lx. pt. ii.) says—"The countries that are freest from excess in drinking are Spain and Italy; if a woman can prove her husband to have been thrice drunk, by the ancient laws of Spain she may plead a divorce from him."

If we may believe Hector Boece, Scotland did early recognise that the procreation of the unfit should be restrained. He recorded: "If any were troubled with the falling evil, or leprosy, or fallen frantic, or otherwise was out of his wits, they were diligently sought out; and, lest those diseases should pass further by infectious generation into their issue and posterity, they gelded the men. But the women were secluded to some odd place far off from the company of men, where if she afterwards happened to be gotten with child, both she and the infant were run through with the lance. Gluttons and ravenours, drunkards, and egregious devourers of victuals were punished also by death, first being permitted to devour so much as they listed." These were Draconian methods which marked the dawn of civilisation, and there are yet those who advocate similar measures. But we must limit our proposals to practical suggestions to modify the liberty of the subject to destroy himself and confer an heritage of woe upon his offspring. Legislative protection is not within the sphere of our activities. True, Dr. Toulouse has formulated a proposal for France, where the consent of parents must be given before marriage, suggesting that the contract of marriage should be protected by an investigation into the physical condition of the parties. He suggests that they should be examined by medical men in the manner incumbent upon those who enter on policies of life insurance. He would declare any marriage void if facts as to morbid heredity were concealed. Certainly such an enactment would direct public attention to the wrong-doing which now proceeds unchecked, but it would bring in its train evils which demand consideration. For instance, how far would the state be justified in branding with illegitimacy the offspring of those to whom marriage was forbidden, thus adding to the burden of their neurotic inheritance social disqualifications which they are specially unfit to bear? Our duty lies nearer at hand, we must hope to stem the tide of degeneracy and insanity by our efforts to educate the nation and to guide it in proper directions. Our present restrictive legal enactments fail by reason of their incompleteness, for the inefficacy of the Habitual Drunkards Acts and the Contagious Diseases Acts is deplorable not only in regard to the present generation, but still more in regard to those who are to come.

LITERATURE.—The leading works of reference for the etiology, symptomatology, and treatment are given on page 402.

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PATHOLOGY

THOSE disorders of the nervous system to which collectively the terms "mental diseases" and "insanity" are commonly applied, constitute a clinical and not a pathological group. They are placed together because all of them in part manifest themselves by derangement of the highest functions of the brain. But a pathological classification cannot depend upon symptomatology. It must be based chiefly upon etiology and pathological anatomy. To pathology, therefore, "mental diseases" are merely diseases of the nervous system. Notwithstanding the rapid advances that have been made during recent years in our knowledge of the morbid changes that affect this portion of the human organism, so very much still remains entirely obscure that a complete identification and precise classification of the various diseases to which it is subject are as yet impossible. Although mental diseases do not form a special pathological group, the separate consideration of their pathology is amply justified in view of the great practical importance of the clinical division, and the fact that the ultimate aim of pathology is to aid the clinician in his battle against disease.

The etiology of insanity has already been fully dealt with in a previous article (p. 292). It remains to consider here (A) the nature of the morbid processes upon which insanity depends, or its pathogenesis, and (B) its pathological anatomy.

A. PATHOGENESIS

"Heredity and stress," or the predisposing and the exciting causes of insanity, are the two great factors of which we have to endeavour to understand the mode of action.

The vast importance of the part played by "an inherent faulty organisation of the individual" in the production of insanity does not admit of dispute at the present day. But some authorities, I think, go too far when they assert that without faulty heredity there can be no insanity. For example, it has been stated that "the difference between the person who may become insane and the person who will not become insane is one entirely of hereditary predisposition."[1] Whether this statement is true or false depends largely, but not exclusively, upon the extent to which we restrict the meaning of the term "insanity." For example, a person suffering from hemiplegia may not be regarded as insane, but nevertheless he may present a much greater amount of disorder of his higher cerebral functions than many persons who are so regarded. But even within the limits of technical insanity there are some cases from the pathology of which hereditary predisposition to mental disease may be readily excluded. A clear instance is to be found in that form of syphilitic insanity which is associated with endarteritis obliterans of the cerebral arteries. In such cases the mental derangement arises simply from interference with the nutrition of certain of the cortical nerve-cells that subserve the associative or intellectual functions, and is essentially independent of, although in certain instances it may be intensified by hereditary predisposition to insanity. Although a faulty heredity cannot, therefore, be admitted to be an absolutely constant factor in the production of insanity, it certainly plays an essential part in the pathogenesis of the vast majority of cases. The questions of its exact nature and mode of operation have given rise to endless speculation, but are still extremely obscure, and scarcely call for discussion here. It will be sufficient if we bear in mind the facts that a hereditary predisposition to mental disease unquestionably exists, that it is extremely common among all civilised races, and that it is specially liable to betray its presence at certain epochs in the life of the individual, namely, those of puberty, adolescence, the climacteric and senile involution.

While a certain comparatively small number of cases of insanity do not require the postulation of any hereditary predisposition in order to explain their pathogenesis, there is, on the other hand, another, somewhat larger class, in which the exciting causes of insanity do not play any essential part. Such cases arise simply from special aberrations of ontogenetic evolution; they constitute one of the end products of degeneration of the race. They are represented by a certain proportion of idiots and imbeciles, and by that class of cases commonly included under "the insane diathesis."

The various possible exciting or determining causes of insanity are exceedingly numerous, and are variously classified. But recent researches have conclusively demonstrated the paramount importance of those that are of a toxic nature. Indeed the exciting causes of insanity that can now be excluded from this category are extremely few, being represented by little more than traumatism and some of the non-traumatic gross lesions. Even such conditions as worry and anxiety, emotional excitement, excessive mental work, loss of sleep and physical exhaustion, which are generally recognised as being fertile causes of insanity, are now believed to produce their effects mainly through the alterations in metabolism and consequent auto-intoxication that they entail.

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[1] John Macpherson, *Mental Affections*, 1899.

The toxic agents that determine the occurrence of insanity in predisposed persons may be (1) introduced from without; (2) they may be formed within the body in the course of various infective and non-infective diseases; (3) they may be generated in consequence of various disorders of metabolism (*auto-intoxication*); (4) they may arise in the contents of the gastro-intestinal canal and be absorbed into the system (*auto-intoxication from the gastro-intestinal canal*); and (5) they may be developed by the action of organisms which have gained admission from the alimentary tract into the blood-stream (*auto-infection*). As corresponding typical examples of forms of mental disease determined by the action of toxines coming to act upon the body in these various ways, the following may be mentioned: (1) alcoholic insanity; (2) syphilitic and rheumatic insanities; (3) diabetic insanity; (4) epileptic insanity; and (5) probably certain cases of acute mania.

Simple auto-intoxication may be brought about by many different conditions, including the defective functional action of various glandular organs (*e.g.* the liver, thyroid, and kidneys), and "physiological instability," in consequence of which the balance between waste and repair is not perfectly maintained. There can be no doubt that such abnormal conditions specially tend to arise in certain individuals owing to particular forms of constitutional weakness. Auto-intoxication from the gastro-intestinal canal has in recent years been shown to be a very common and highly important determining cause of insanity.

As normal mental action is solely the expression of the functional activity of certain neurons or nerve-cells within the brain, the toxic agents which determine the occurrence of mental disease can do so only in consequence of some injurious influence which they have upon these neurons. This injurious influence may be exerted either directly or indirectly. In the former case the poisons are carried to the nerve-cells by the blood-stream, or, more rarely, generated in the lymph by which these tissue-elements are bathed. They produce their indirect influence in various different ways. They may set up morbid changes in the walls of the cerebral blood-vessels, in consequence of which the nutrition of the nerve-cells is affected; they may cause general anæmia, with a similar result; it is probable, also, that they are capable of producing disorders of the cerebral circulation, more especially general or local active congestion, which likewise may disturb the functional activity of the nerve-cells; and, finally, they may cause structural changes in the walls of the channels which convey away the cerebro-spinal fluid, in consequence of which the outflow of this fluid is obstructed, the lymphatic circulation of the brain is deranged, and the lymph becomes charged with the waste products of metabolism which poison the cerebral tissues.

The alterations that occur in the brain and its envelopes from these causes in mental diseases have already to a certain extent been elucidated, and their special relationship to insanity amply proved. Some of them are briefly described in the next subsection.

We can now in general terms explain the occurrence of the large majority of the different forms of mental disease as the direct or indirect result of the action of toxic agents upon cerebral tissues of hereditarily weak resistive power, with, however, the reservation that it is at least possible that in some instances the hereditary defect lies not in the brain, but in other organs, which in consequence thereof are liable under certain circumstances to derangements of function which result in the formation of toxines of special virulence to the nervous tissues. But in regard to the more precise details of the pathological processes upon which the various phenomena of insanity depend, only a few facts are as yet very clearly understood. The pathology of insanity is a subject of very great complexity and difficulty. Its progress has necessarily been extremely slow in comparison with that of most other departments of pathological science. It has had to wait long for the foundation of normal anatomy, physiology, and experimental pathology, upon which alone it can be based. Indeed, it can be said to have received such a foundation only within the last three or four years. For this great advance science is indebted to a very large number of workers, but in a special manner to Lugaro, Marinesco, van Gehuchten, and Ramon y Cajal.

Although the present state of knowledge does not render it possible to explain the *exact* pathogenesis of any single form of insanity, recent investigations have led to the formulation of several highly important physiological conclusions, some of which throw a large amount of light upon certain of the special phenomena of mental disorder. As is now well known, Flechsig has lately mapped out the brain into sensory and associative (or intellectual) centres, and has attributed special functional values to certain portions of the latter. If these conclusions could be regarded as definitely proved, they would serve as a basis for the explanation of various phenomena of insanity. But it happens that, at the present moment, the very foundations of Flechsig's work are being hotly assailed by several other high authorities upon such questions, and while his views are thus still in the region of controversy, it is impossible to use them as established facts of cerebral physiology. Of much greater immediate value are, I think, some of the conclusions upon the physiology of the neuron, recently formulated by Lugaro, more especially his theory of the contractility of the gemmulæ of the protoplasmic processes, and his hypothesis regarding the distinctive functional values of the intra neuronic and inter neuronic elaborations.

On the basis of certain experimental observations, Lugaro[1] maintains that the gemmulæ of the protoplasmic prolongations of the nerve-cells (which, it is now very generally believed, receive the nervous stimuli from the terminations of the axis-cylinder processes of other nerve-cells) are capable of undergoing rapid movements of retraction and expansion which have an important functional significance. He thinks that during sleep there is general expansion of these gemmulæ, implying multiplication of contacts, and wide diffusion and consequent weakening of stimuli. In conditions of fatigue their contractility is greatly diminished. After rest it is fully restored. During psychical activity they undergo continuous movements of retraction and expansion, whereby contacts with the terminal processes of other nerve-cells are broken and made. He believes that a nerve-cell, on receiving a stimulus, must while elaborating it retract all its drawbridges in order to prevent the access of other stimuli, and that such modifications must occur in a very large number of neurons in correspondence with every elementary state of consciousness. The retraction does not, however, last longer than the said act of consciousness, the gemmulæ quickly reassuming their condition of expansion and readiness to receive new impressions.

In accordance with this theory, we may conceive of the insomnia so commonly associated with mental diseases as dependent upon an irritable condition of the cortical neurons, in consequence of which the gemmulæ of their protoplasmic prolongations are unable, even in states of fatigue, to remain in their position of rest (*i.e.* general expansion), but continue to contract in response to the slightest stimulus. We may conceive of stupor as dependent, on the other hand, upon an abnormal condition of the cortical nerve-cells, in consequence of which the contractility of their gemmulæ is more or less completely abolished.

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[1] *Rivista di patologia nervosa e mentale*, 1898, f. 8.

The other hypothesis referred to is also supported by experimental evidence of the strongest kind. Lugaro[1] maintains that two distinct elaborations of the external impressions occur in the nerve-centres, the one interneuronic, at the terminations of the afferent fibres, and the other intraneuronic, between the wave conveyed by the dendrites and the dynamical processes that develop in the interior of the cell-body; and that the first corresponds to the phenomena of consciousness or perception (*fenomeni di conoscenza*), the second to the affective states. He further summarises his thesis as follows: The external impressions arrive at the pericellular and peridendritic arborisations, and there assume relations corresponding to the objective relations; whence the perceptive act. The product elaborated and received by the dendrites reaches the cell-body, in which a new effect is produced, a subjective modification in accordance with its state of nutrition and specific constitution; whence the affective act. According to the character that the process of intracellular elaboration assumes, one has finally various forms of discharge from the cellular body by way of the axon.

Regarding the relation of this hypothesis to questions of neuropathology, I may perhaps be permitted to quote views that I have already expressed elsewhere:[2]—

"In considering the bearing of this hypothesis upon problems in pathology it is, I think, important to have clearly in mind that an interneuronic elaboration may be suppressed by interruption of the conducting fibrils at any point in their course in one or other of the two neurons concerned, and not merely by some abnormal condition of the intervening lymph. The former is probably by far the more common cause of such suppression in mental disease. The latter would appear to be the essential factor in chloroform anæsthesia and allied conditions. We may probably exclude any state of continued general contraction of the gemmulæ from the possible, or at least the common, causes of suppression of the interneuronic discharge, for contraction of these appendages would seem to be essentially associated with their functional activity; it is the response to a stimulus received, and is of short duration; the position of rest is that of expansion.

"Upon this hypothesis we may conceive of states of simple mania and melancholia as dependent upon abnormal metabolism within the substance of the nerve-cell, and understand how they are compatible with unimpaired action of some of the purely intellectual faculties, the integrity of which is dependent upon the maintenance of certain mutual relations of groups of neurons. We may picture a momentary amnesia as implying a temporary failure of a particular interneuronic discharge, profound dementia as dependent upon the fact that the majority of the special interneuronic connections that education had slowly developed and perfected have suffered destruction, owing to the death of one or both of the neurons concerned with each. Between these two degrees of failure of mental action we may conceive of endless varieties, dependent upon the number and the special functional rôle of the neurons involved. In a general way we may regard a delusion, or at least some forms of delusion, as the result of suppression of certain interneuronic elaborations included in those that are collectively required to form a particular association. In consequence of this suppression of one or more of its usual factors, the association is rendered imperfect, the mental picture is distorted, one part of it is lost to view, and another attains undue prominence, just as a little flaw in an otherwise faultless window pane is capable of making us see valleys and hills in a landscape in which there are none. The suppression of the interneuronic discharge being purely of local incidence, it may be inferred that it does not depend upon any abnormal condition of the intervening lymph, but upon some alteration in the fibrils of the neurons concerned. Such alterations commonly imply involvement of the whole cell-element and a lesion of considerable severity. It is known that for every nerve-cell that suffers to this extent from any toxic condition there are generally several others that suffer in a smaller measure. From these considerations we may perceive at least one reason why delusions are generally accompanied by either excitement or depression. It is further to be observed, that the suppression of the interneuronic discharge is not necessarily permanent; it may result merely from fatigue of an element of which the structural and functional integrity is already impaired, or from a degenerative process which may be arrested on withdrawal of its toxic cause."

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[1] *Riv. di patologia nerv. e ment.*, 1899, f. 11, 12.

[2] *Text-book of Pathology in Relation to Mental Diseases*, 1900.

Morbid Conditions of the Cerebral Circulation.—Alienists have long believed that simple disorders of the cerebral circulation, more especially general or local congestion and anæmia, play a very important part in the pathogenesis of various forms of mental disease. This has seemed to them the inevitable teaching not only of their clinical experience, but also of the phenomena that they have observed, times without number, in the asylum post-mortem room. Recently, however, its accuracy has been seriously called in question by the results of the experimental observations of Leonard Hill[1] upon the physiology and pathology of the cerebral circulation. On the ground of these investigations, the high scientific value of which cannot be denied, Hill concludes that the volume of blood in the brain is in all physiological conditions but slightly variable (the Monro-Kellie doctrine), that, consequently, an increase of blood in the cerebral arteries implies diminution of the amount in the veins, and *vice versa*, that there is no general or local cerebral vaso-motor mechanism, that, although the nerve-fibres demonstrated in the pial arterioles may be vaso-motor in function, "their power must be insignificant, since in every experimental condition the cerebral circulation passively follows the changes in the aortic and vena cava pressures," that "the cerebral circulation is controlled by the vaso-motor centre acting upon the splanchnic area," that "in all physiological conditions a rise of arterial pressure accelerates the flow of blood through the brain and a fall slackens it," that "arterial hyperæmia of the brain produces no experimental results of importance," and that "cerebral venous congestion, on the other hand, is of great pathological significance," mainly because it implies arterial anæmia. He summarises his grounds for agreeing with the Monro-Kellie doctrine, as follows: "When the arterial pressure rises, the expansion of cerebral volume can take place only to a certain limited amount.

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[1] *The Physiology and Pathology of the Cerebral Circulation*, London, 1896. "The Experimental Pathology of the Cerebral Circulation," Clifford Allbutt's *System of Medicine*, vol. vii. 1899.

For as soon as all the cerebro-spinal fluid has been driven out from the cranium the brain is everywhere in contact with the rigid wall of the skull. The cerebro-spinal fluid in the cranium of the living animal is insignificant in amount. Any further expansion of the arteries and capillaries can now only take place by an equivalent compression of the veins, for the semi-fluid brain matter is incompressible. The reservoirs of blood in the veins will therefore be so far constricted until the cerebral venous pressure again becomes equal to the intracranial pressure; that is, to the pressure of the brain against the veins. Then the whole circulatory system of the brain will have assimilated itself to a scheme of rigid tubes."

At the present day, in this country at least, Hill's investigations are generally looked upon as having virtually closed the discussion upon this particular question of the variability of the quantity of blood within the cranium. Most of his other conclusions, stated above, are also very widely accepted as accurate.

I have recently[1] endeavoured to show that much of Hill's teaching upon these subjects is untenable, more especially his homologation of the Monro-Kellie doctrine and his dictum of the absolute control of the cerebral circulation by the vaso-motor centre acting upon the splanchnic area. In the space available here it is impossible for me to explain fully my reasons for taking up this position. The reader who may think them worth examining will find them stated at length in the work above referred to. With regard to the question of the variability of the quantity of blood within the brain, I contend that Hill has been led into false conclusions by failing to take into account the variability of the quantity of fluid in the lymph-spaces within the brain, and the elasticity of the cerebral vessels and nervous tissues, and by accepting as an axiom what can be shown to be really one of the most transparent of fallacies, namely, the doctrine of the incompressibility of the brain; and that, with regard to the question of the control of the cerebral circulation, his experimental evidence is by no means conclusive, being contradicted by the results obtained by other observers, whose methods of investigation were no less trustworthy. I maintain that, when all the known facts bearing upon the question are taken into consideration, there are the strongest reasons for believing that the mechanism by which the cerebral blood-supply is regulated is not nearly so simple as Hill would have us believe, and that the case for the existence of a purely local mechanism is now an exceedingly strong one.

According to those who believe in the accuracy of the Monro-Kellie doctrine, active congestion of the brain can only consist in an increase in the rate of flow through the cerebral blood-vessels; passive or venous congestion must be associated with diminution in the rate of flow and with compression of the capillaries and arterioles, and therefore with cerebral anæmia; cerebral anæmia cannot imply diminution in the quantity of blood in the brain, but merely retardation of the flow.

I hold that such teaching is quite erroneous, for, as originally maintained by Burrows in 1846, and as is confirmed by recent experimental and histological observations, variations in the quantity of blood in the cranial cavity are rendered possible within considerable limits by inverse variations in the quantity of cerebro-spinal fluid, contained not only in the pia arachnoid, but also in the lymph-spaces of the brain itself. Active congestion of the brain, therefore, implies far more than mere acceleration of the blood-flow; it is accompanied by distension of the arterioles and capillaries, and it may be also of the veins, and an important increase in the amount of blood within the brain. Similarly, a true general passive congestion of the brain is possible; distension of the veins does not necessarily entail compression of the capillaries and arterioles and consequent arterial anæmia. On the other hand, cerebral anæmia does not necessarily consist merely in a retardation of the blood-stream, but may manifest itself as an actual diminution in the quantity of blood in the brain. Under conditions of high intravascular pressure, distension of the cerebral vessels is not limited by the rigid cranial walls and the "incompressible brain substance," as Hill virtually asserts, but essentially by the strong elastic walls of the vessels themselves.

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[1] *Loc. cit.*

Although there are still many difficulties in the way of determining the exact state of the cerebral circulation in mental diseases, there appear to me to be sufficient grounds for the following conclusions:[1]—

General paralysis is constantly attended with active congestion of the brain, which, however, specially affects the cerebral cortex. In the third stage this congestion often reaches an extreme degree, and is then accompanied by a loss of elasticity of the capillary walls, in consequence of which they do not contract after death. Hence the cortex appears more or less deeply congested, often in a patchy manner.

Senile mania is associated with cerebral hyperæmia, which tends to affect particular areas specially. The incidence and distribution of this hyperæmia are probably in large part determined by organic changes in the vessel walls.

In *acute insanities* there is always hyperæmia of the cerebral cortex. In some cases, however, there is no distinct post-mortem evidence of such hyperæmia. It is probable that in these the dilated vessels have contracted after death owing to the fact that the elasticity of the capillary walls has not been greatly impaired. In many other cases there is to be observed very distinct patchy congestion of the cortex, which may be interpreted as indicating that the elasticity of the capillary walls has been impaired to a greater extent in some areas than in others. In a large percentage of cases there is seen a condition of intense general congestion of the cortex. There is, further, sometimes to be observed at autopsy a condition of intense congestion throughout the whole encephalon, accompanied generally by multitudes of minute capillary hæmorrhages. This condition corresponds to what has been described as "hæmorrhagic encephalitis." In delirium tremens, which may be included among the acute insanities, there is post-mortem evidence of extreme general hyperæmia of the brain, with commonly very extensive permanent distension of the capillaries.

In *choreic insanity* there is always well-marked hyperæmia of the whole brain.

There are the strongest reasons for believing that such cerebral hyperæmia in mental diseases is essentially only a part of a complex pathological process, and that it is not in itself a primary cause of the cerebral disorder.

The nutrition of the brain in common with that of other organs is undoubtedly seriously affected by general anæmia, whether of a quantitative or a qualitative character. Certain kinds of hereditary instability and feeble resistiveness of the brain appear to be specially liable to manifest themselves under such conditions of malnutrition. It is probable that to such concurrence of pathological factors a large number of cases of chronic melancholia, as well as of some other forms of chronic insanity, may rightly be attributed.

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[1] *Loc. cit.*

B. PATHOLOGICAL ANATOMY

The Scalp.—Morbid adhesion of the epicranial aponeurosis to the pericranium is present in about 50 per cent of persons dying insane. An extreme degree of this adhesion, such as to render detachment of the scalp impossible except by dissection, occurs in over 10 per cent of cases. The condition is most common in general paralysis and in epilepsy. In a large majority of instances it is associated with either thickening or condensation of the bones of the skull.

"Hypertrophy of the scalp" is a rare abnormality which appears to have been observed, at least in a well-marked form, only in the mentally defective, and in the majority of instances in genetous idiots. It consists in an abnormally redundant, thick, lax, and mobile condition of the integument of the upper and posterior parts of the head, which at the same time presents a number of roughly symmetrical, generally antero-posterior and transverse furrows and ridges.

Hæmatoma auris, which is exceedingly common in the insane and comparatively very rare in other persons, is described in vol. iv., p. 262.

The Skull.—Morbid conditions of the skull consist of two distinct classes, namely (1) variations in form and size overstepping the limits of the typical or normal, and (2) changes in the structure of the bone, evidenced by abnormalities in thickness, consistence, and histological characters.

The skulls of the insane in this country have not yet been sufficiently studied by strict craniological methods to allow of precise statements being made regarding the special morphological features that they exhibit. Only a few of the more distinct abnormalities that have been observed need be mentioned here.

The microcephalic skull, which exhibits a characteristic form as well as an abnormally small size, has, according to the observations of Sir George Humphry,[1] an average circumference of 16·7 inches. This authority has described its chief characters as follows:—"The brain-case is small or contracted in all its dimensions, in the base as well, though less than in the vault; and it is most so in the fore part, the frontal bone being sloped back and narrow, with deep temporal fossæ behind the external angular processes, and with curved orbital plates, which narrow the ethmoidal fossæ. The parts in the interior are contracted and often thickened."

The macrocephalic or hydrocephalic skull has its greatest circumference above the glabella and occipital point. The authority just quoted has described its chief characters as follows:—"The brain-case is expanded in all directions, to some extent in the base, but much less than in the vault. The forehead bulges forwards, beetling over the face; the orbital plates are in the young flattened and pressed down, and the superciliary ridges are elevated and depressed. The temporal fossæ are shallowed, and the occipital condyles are not prominent."[2]

Although asymmetry of the cranium is not necessarily an indication of an abnormal brain, high degrees and certain special forms of it are certainly far more common in the insane than in the mentally sound.

A special type of deformity, to be observed in some cases of idiocy and imbecility, has been termed *crania progenea.* It consists in abnormal prominence of the lower jaw and forehead, defective development of the occipital region, and narrowing of the face.

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[1] *Journ. Anat. and Physiol.* vol. xxix. 1895, p. 307.

[2] *Journ. Anat. and Physiol., loc. cit.*

Another abnormal form of cranium has been termed by Clapham and Clarke "the insane type," having been found only in cases of insanity. Its special feature consists in the greatest transverse diameter being in its anterior third instead of behind the middle point.

Absence of the normal overriding of the dentary arcades, which the researches of Sir William Turner[1] have shown to be an atavistic character, has been found to be much more common in the insane than in the mentally sound.

Most authorities upon mental diseases at the present day regard deformities of the hard palate as an important sign of a bad initial neurotic heredity. The accuracy of this view is, however, strongly contested by certain other authorities upon such subjects, and therefore remains in question.

Alterations in the thickness, texture, and weight of the cranial bones are exceedingly common in the insane. The calvarium is generally affected to a much greater extent than the base. Distinct thickening may be observed in about 50 per cent of cases examined after death. The change may be local or general, and may be combined either with osteosclerosis or osteoporosis. It is most common in cases of general paralysis and senile insanity. Atrophic thinning of the skull also occurs, but is comparatively rare. Most of the examples are found in cases of senile insanity. Condensation of the bones of the skull, or osteosclerosis, is present in about 30 per cent of all cases. The condition is practically a constant one in general paralysis. It is accompanied by thickening in the majority of instances. General softening of the bones of the skull, or osteoporosis, has been observed to occur in about 5 per cent of cases.

The cause of these alterations in the texture of the bones of the skull in cases of mental disease is probably the same as that which leads to the changes in the dura mater presently to be described.

The Dura Mater.—Morbid adhesion of the dura to the skull is common in various forms of insanity. It has been found by different observers in from 15 to 44 per cent of cases. The condition is probably in most instances related to the circumstance that the periosteal surface of the membrane is depositing new bone. Morbid adhesion to the pia arachnoid is comparatively rare, and is almost always of small extent. It is probably of inflammatory origin. General thickening of the dura is very common in the insane, but is somewhat difficult to recognise with the unaided eye. It depends upon the occurrence of a slow sclerotic process, the evidence of which can be readily observed on microscopic examination. Atrophic thinning of the membrane is very occasionally met with, mostly in cases of senile insanity. Small bony nodules are sometimes present, either in the substance of the dura or on its inner aspect, most commonly adhering to the falx. In most instances they probably represent merely a developmental anomaly in a membrane of which one surface is a periosteum. Inflammation of the dura mater has little or no special relationship to insanity. The so-called *pachymeningitis hæmorrhagica interna* can be shown to be of different origin. On the other hand, degenerative changes are exceedingly common, and have considerable pathological importance. Subdural false membranes can be recognised to be present in about one-fourth of cases examined post-mortem. They vary greatly in size, appearance, and consistence in different instances. They may be limited to a small portion of the dura, or extend over the entire surface of the cranial and spinal membranes. When they are large, and at the same time somewhat localised, they are generally cystic in the centre.

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[1] *Journ. Anat. and Physiol.*, 1891, July.

They are always more or less adherent to the dura, almost never to the pia arachnoid. Their pathology has given rise to a long-standing controversy, which has been chiefly concerned with two opposing theories regarding their origin, namely, the inflammatory and the hæmorrhagic. According to my own observations two classes of subdural false membranes require to be distinguished. One is the result of a simple hæmorrhage into the subdural space, most commonly from a pial vein. The other depends upon certain degenerative conditions of the dura, which are exceedingly common in cases of chronic insanity, more especially general paralysis and senile insanity. In order that we may be able to appreciate fully the nature and importance of these degenerative changes, it is necessary to understand that the dura mater contains innumerable minute lymphatic channels which pass from its inner surface and discharge into the veins both in the membrane and in the cranial bones. These channels convey away the cerebro-spinal fluid. At the inner aspect of the membrane, immediately underneath the surface endothelium, there are very numerous large capillaries, each of which lies in one of these lymphatic channels, which in this position are generally referred to as the perivascular canals of the dura. The walls of these channels are nourished chiefly by the cerebro-spinal fluid which flows through them. In many forms of mental disease this fluid has distinctly toxic properties, owing mainly to the disordered metabolic changes going on in the cerebral tissues, but partly also to general toxic conditions. Hence the endothelial cells of the dural perivascular canals are irritated and imperfectly nourished. They consequently undergo proliferative and degenerative alterations, which result in compression and obliteration of the superficial capillaries. At the same time general proliferation of the dural tissues tends to occur. Obliteration of the original superficial capillaries is constantly followed by the throwing out of new capillaries on the surface of the membrane. These new vessels tend to give way under various circumstances, but especially during the last few days of life in all cases in which the patient sinks slowly. The great majority of the subdural false membranes that are to be observed in the insane are developed as the result of hæmorrhages brought about in this way. With few exceptions they have the histological characters of recent formations. They are essentially composed of blood-clot, which, however, is commonly in course of being vascularised. Associated with these pathological processes there are various other proliferative and degenerative changes in the tissues of the dura mater leading to the formation of surface granulations, hyaline concentric bodies, mulberry bodies, etc.

The Pia Arachnoid.—The leptomeninges are composed of white fibrous tissue, or perhaps of a special form of elastic tissue, which is arranged in such a way as to form a spongy lymph sac. This structural arrangement extends throughout the whole of the membrane, which, upon this and other grounds, Dr. James Middlemass and I have contended, contrary to the general teaching, should be regarded as a single structure. I have recently endeavoured to give a full statement of the case in support of this view.[1] It is important to understand that the pia arachnoid is not directly nourished by the vessels it contains. These are simply passing to and from the cerebral tissues. The membrane depends for its nutrition upon the lymph or cerebro-spinal fluid which, escaping from the capillaries of the brain, passes along the adventitial lymph-channels of the intra-cerebral vessels, enters the arachnoid spaces, and passes across the subdural space into the perivascular canals of the dura mater.

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[1] *Loc. cit.*

The most important pathological alteration that affects the pia arachnoid in cases of insanity is one which presents itself as milkiness, thickening, opacity, and other related abnormal appearances. Over 70 per cent of cases of insanity show more or less pronounced evidence of these changes on post-mortem examination. The morbid process is essentially the same as that which occurs on the opposite side of the subdural space, and has the same cause, but differs in respect of the fact that it is not complicated by the vascular changes which occur in the dura. The endothelial cells lining the arachnoid spaces and the outer surface of the membrane undergo proliferative and degenerative changes. At the same time a slow hyperplasia of the connective tissues takes place. Hence the membrane increases in thickness and assumes a milky appearance.

The Cerebral Blood-vessels.—These present several special anatomical features which it is necessary to understand in connection with the study of the pathological changes to which they are subject. All the blood that they convey has to pass (with one or two immaterial exceptions) through the nervous substance. The pia arachnoid has no capillary circulation of its own, and hence there is no possibility of short-cutting of the cerebral blood-supply. The small pial arterioles anastomose somewhat freely with each other. The cerebral capillaries are peculiar in that they possess an external coat. This is composed of highly elastic connective tissue fibrils continuous with those of the adventitia of the arterioles and venules. A special set of capillaries, coming off from the pial arterioles, supplies the first layer of the cortex. All the intra-cerebral vessels have normally in their walls a small amount of a pale yellow substance which increases with age.

One of the most important morbid changes to which the cerebral vessels are subject is the hyaline fibroid degeneration of Gull and Sutton. It consists in hyaline swelling, or of fibroid thickening, of the connective tissue fibrils of the adventitia. The first represents the degenerative and the second the reparative phase of the morbid process. This form of disease occurs in arterioles, capillaries, and venules. It specially tends to affect the capillaries of the first layer of the cortex, which in senile insanity are often extensively obliterated by it. Another exceedingly important vascular change in cases of chronic insanity is endarteritis deformans, or atheroma. It affects not only the large cerebral arteries, but also the small arterioles and capillaries within the brain. It leads to narrowing of the vascular channels, and not infrequently to their obliteration. Both this vascular alteration and hyaline fibroid degeneration lead to the formation of atrophic softenings. The pathology of endarteritis deformans is still in dispute, but, on the evidence of the changes that may be observed in the cerebral vessels, the disease appears to depend upon an abnormal condition of the blood, in consequence of which the tissues composing the intima are injuriously affected in their nutrition, and undergo either proliferative or degenerative changes. With the temporary cessation of the nutritive disturbance reparative processes occur, resulting always in some increase in the thickness of the intima. The degenerative phase is constantly accompanied by weakening of the muscular coat of the arterioles, and, if associated with high blood-pressure, is apt to lead to local dilatation and the formation of miliary aneurysms. In correspondence with the special frequency with which their cerebral arterioles are affected by atheroma, the insane are specially prone to the occurrence of cerebral hæmorrhage, which is connected in most instances with the rupture of a miliary aneurysm, or of an arteriole otherwise weakened by this form of morbid change. Endarteritis obliterans affects both the large and the small cerebral arterioles in the vascular form of syphilitic insanity. Acute periarteritis of the intracerebral arterioles is an almost constant change in the advanced stages of general paralysis. Colloid degeneration and calcification have occasionally been observed in cases of insanity, but are certainly rare.

The Neuroglia and Mesoglia.—The question of the normal structure of the neuroglia has given rise to much discussion. I have recently contended that the tissues to which this name has hitherto been applied consist really of two totally different cellular elements, the one of which is epiblastic in origin and the other mesoblastic.[1] The evidence in support of this view consists mainly (1) in the fact of the differentiation of the latter tissue-element by means of the platinum method; and (2) in its harmony with the recent important observations of Capobianco and Fragnito[2] upon the histogenesis of the neuroglia. The true neuroglia consists of cells with numerous, only very slightly branched, specially differentiated processes, at least many of which are attached to the vessels and other structures. The mesoglia in its typical form consists of cells with numerous processes, which branch dichotomously, and are never attached to the vessels or to other structures. The two kinds of cell can be shown to behave quite differently in pathological conditions. The neuroglia serves to support the nervous elements, and it is the tissue of repair in the brain. There is conclusive evidence that the mesoglia cells do not subserve either of these functions. Their rôle seems to correspond pretty closely to that of connective tissue corpuscles in other organs. It is at least certain that they are capable of acting as phagocytes.

In conditions of chronic irritation in the brain, as in general paralysis, choreic insanity, and chronic alcoholism, there is usually well-marked hypertrophy and proliferation of the neuroglia in certain parts of the brain. Similar changes occur wherever the nervous tissues have been extensively destroyed, as for example in the vicinity of the atrophic softenings so common in cases of senile insanity. The mesoglia cells form a certain proportion of the granular corpuscles (others being leucocytes) which commonly occur wherever dead cerebral tissues are being absorbed. In some irritative conditions they tend to proliferate and to degenerate into amyloid bodies.

The Cortical Nerve-cells.—In recent years very remarkable advances have been made in our knowledge of the structure of the nerve-cell. Two chief classes of nerve-cells are now generally distinguished, namely the *somatochrome*, which contain chromophile substance in their protoplasm, and the *karyochrome*, which are generally much smaller and do not contain this substance in their protoplasm. The somatochrome nerve-cells are by far the more important, and they alone need be dealt with here. The nucleus possesses a distinct limiting membrane, a reticulum and granules, and a single nucleolus. All of its structural elements are acidophile in reaction, with the exception of three or four small particles adherent to the nucleolus, which are basophile, and which constitute the whole of the chromatin in the cell. The protoplasm surrounds the nucleus, and extends on the one hand in the form of long-branching and gradually tapering processes, termed the protoplasmic processes or dendrites, which at some little distance from the cell-body are clothed with minute, often filiform, appendages, the gemmulæ or thorns; on the other hand it extends in the form of a single process, termed the axis-cylinder process or axon, which at a short distance from the cell-body generally gives off some delicate branches, the collaterals. The main process may pass into a medullated nerve-fibre, or run a comparatively short course as a non-medullated fibre. The dendrons are organs of reception of the nervous impulses, the axon the apparatus concerned with their discharge. The protoplasm contains innumerable minute fibrils, which are now generally regarded as the conducting element of the nervous impulses. These fibrils course for the most part independently in the processes, but form a network in the cell-body. The interstices of this network contain localised aggregations of a substance which presents a strong affinity for certain dyes, such as toluidin blue. These aggregations constitute the Nissl-bodies or chromophile particles of the nerve-cell protoplasm. There is generally a collection of a pale yellow substance, the nerve-cell pigment, in the vicinity of the nucleus. Several minute lymph canaliculi course within the body of the larger cells and open upon the surface. They were first observed by Golgi, who described them as a special endocellular reticulum.

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[1] *Loc. cit.*

[2] *Annali di Neurologia*, 1898, f. 2, 3.

The neuron theory, according to which each nerve-cell (with its processes) is anatomically and physiologically an independent element, has lately been boldly attacked by Apáthy, Bethe, and Nissl, but, nevertheless, retains its place as a canon of neurology. It is still defended and maintained by such high authorities as Lugaro, Marinesco, van Gehuchten, Ramon y Cajal, Barker, and Lenhossék.

It has now been definitely proved that the chromophile substance of the protoplasm is utilised during the functional activity of the nerve-cells.

Recent experimental investigations have thrown a great amount of light upon the meaning of many of the abnormal appearances to be observed in the nerve-cells of the human subject. It has been shown that the nerve-cell is exceedingly sensitive to the action of many different toxic substances, and that it undergoes definite structural alterations after injury to its axon. The pathological process set up by the former cause has been designated *primary degeneration*, and that resulting from the action of the latter *secondary degeneration*. In primary degeneration the changes vary considerably in accordance with the toxine employed and the intensity of its action. In general the condition manifests itself first by disintegration of the chromophile substance of the protoplasm (*chromatolysis*), and afterwards by disintegration of the fibrils, degeneration of the nucleus, and death of the whole element. If the alteration does not proceed further than simple chromatolysis, it may be recovered from. In secondary degeneration the alteration begins with disintegration of the chromophile substance in the neighbourhood of the cone of origin of the axon, and gradually extends to the rest of the cell-body. At an advanced stage the nucleus is often displaced to the periphery, and the primitive fibrils of the protoplasm destroyed. The cell may undergo repair, or it may atrophy and disappear.

Primary and secondary degeneration are the only diseases of the fully developed nerve-cell that are at present definitely known. Further research will doubtless enable us to recognise various pathologically distinct varieties of primary degeneration. The many types of morbid change that may be observed to affect the nerve-cells of the human cerebral cortex are to be regarded as indicative of the occurrence of one or other of these two forms of degeneration, and must not be looked upon as in themselves distinct diseases of the cell. Among these types of morbid change the most common are chromatolysis, pigmentary or yellow globular degeneration, vacuolation, varicose atrophy of the protoplasmic processes, varicose hypertrophy of the axis-cylinder process, and displacement of the nucleus.

The following are some of the more important facts that seem now to have been definitely established regarding the pathological alterations in the cortical nerve-cells in different forms of mental disease. In cases of acute mania and acute melancholia dying in the course of the disease, from 25 to 50 per cent of the cells show distinctly marked degeneration of the primary type. This percentage is very much higher than that which is generally to be found in persons succumbing, for example, simply to pneumonia, or exhaustion from any wasting disease, such as phthisis. Moreover, the intensity of the pathological changes is far greater in cases of acute insanity, and there is satisfactory evidence that many of the cells have completely disintegrated and disappeared. In senile insanity and in general paralysis there is constantly a slow degeneration of the cortical nerve-cells, for the most part of a primary type. The number of cells involved is very much larger than that affected in normal senility, and the severity of the alterations is far greater. In general paralysis considerable areas of the cortex are often practically depleted of nerve-cells. In cases of choreic insanity there are always extensive, more or less acute, degenerative changes in the cortical nerve-cells, also of a far more severe character than those that occur from simple terminal auto-intoxications. In chronic alcoholic insanity there is very considerable loss of cortical nerve-cells, while a large proportion of those that remain show degenerative changes either of a primary or of a secondary type. Secondary dementia depends essentially upon the fact that a large proportion of the cortical nerve-cells have suffered complete degeneration. In some cases it can be shown that about 50 per cent of them have entirely disappeared.

Congenital Abnormalities of the Brain.—Some of the slighter of these may not infrequently be observed in persons who, during life, did not exhibit any noteworthy mental aberration or peculiarity; nevertheless such slight structural abnormalities (such as atypical arrangement of the convolutions and sulci, absence or imperfect development of various parts of the brain, heterotopia of the grey substance) are far more common in the insane than in the mentally sound. They are especially common in cases of idiocy, imbecility, and epileptic insanity. Some of the more gross congenital defects, such as microcephaly, are quite incompatible with normal intellectual development, while others, such as porencephaly and absence of the corpus callosum, have only very rarely been discovered in sane persons.

Focal Lesions.—Localised gross lesions of the brain are present in a remarkably high percentage of cases of insanity examined after death. The form and degree of mental derangement that they occasion probably depend mainly upon their localisation and the special reactive qualities of the individual brain. In a large majority of instances they are not, however, the only important structural alterations in the organ, being associated with well-marked pathological changes in various other situations.

By far the most common and most important focal lesions are those of vascular origin. They are represented especially by atrophic and hæmorrhagic softenings. The former especially tend to be multiple. Both are extremely common in the senile insane, but are also not infrequently to be observed in lunatics dying in middle life.

Various forms of cerebral tumour are common exciting causes of insanity. Tubercular nodules are also to be observed with some frequency in the cerebral substance. Localised gummata are occasionally present in cases of syphilitic insanity, but appear to be less common than a diffuse gummatous infiltration of the pia arachnoid and adjacent brain substance, which is generally associated with endarteritis obliterans of the cerebral arteries. Extensive localised areas of sclerosis are occasionally met with, especially, but not exclusively, in epileptics. Their origin is still somewhat obscure. A special form of localised sclerosis of the brain, which, like the preceding, occurs especially in epileptic idiots and imbeciles, has been termed *hypertrophic nodular gliosis.* The nodules vary in size in different instances. They are generally multiple and affect especially the cortex. According to the recent observations of Pellizzi,[1] this form of cerebral sclerosis depends upon a defect of development arising at a time corresponding to the formation of the tertiary sulci.

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Insanity, its Nature and Symptoms.

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I. THE RELATION OF MIND AND BRAIN

(1) BIOLOGY OF CONSCIOUSNESS.—The medical faculty, as well as the general public, still labour to some extent under a time-honoured disability in their view of insanity, for which our philosophers are largely to blame. To the philosopher, indifferent to the pedigree of brains, and habitually averse to appreciate the material conditions of thought, the development of personality is interpreted merely as an evolution of consciousness, sometimes consecutive and intelligible, but

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[1] *Annali di freniatria*, 1899, f. 4; 1900, f. 1, 2.

often inconsequent and fortuitous. For the phases of the human mind, subjectively considered, resolve themselves into moments of self-attention, alternating with periods of attention to things other than the self, and without any apparent thread to bind them together. The attention moves from this to that, sometimes by association of ideas, sometimes because of a fact in the environment which it perceives; but very often also without any apparent occasion either in itself or in its circumstance. Every human mind has gaps in it. Alone, in still darkness, things jump into the mind; and, at any moment in which the attention is not following an idea, nor noticing the outer world, irrelevant suggestions occur. Such interruptions or invasions of the mind are only to be explained by a view of personality which relates it to cerebral processes, some of which are determined by self-directed movements of the attention, some as reactions to the experience of the moment, but also some which are automatic. And especially when we consider insanities and allied states we must have recourse to a cerebral interpretation of the facts. Most people still labour under a view of insanity which regards it merely as the lost wanderings, or the transcendent flights, of the soul. That was an orthodox view about a century ago. Insanity is cerebration—the activities of a disordered cortex —and the modes of consciousness which it exhibits are to be explained as due to realms and tracts whose functions are normally subconscious. It must become our habit to think of the mind as conditioned by nervous states, and of personality as determined by inherited cerebral character. Only by an explicit and detailed study of the mutual relations of spirit and brain can we appreciate either normal personality or these interruptions or invasions of it—loss of power in memory, in judgment, or in purpose, occasional lapses to brute states, muscular violence, emotional frenzy—to which the unstable are liable.

The first step towards an appreciation of the cerebral relation of mind is a biological one. We must conceive of our brain-stuff as not only the vehicle of all that is forthcoming in human character, but also as preserving more or less active rudiments corresponding to the unrecognised memories of all animal and other ancestral experience. The human mind is at once the master and the servant of an organ of whose potencies the sane and orderly person realises only a very small fraction, but an organ which, in dissolution, reveals an infinite store of atavistic associations—"instinctive vestiges," as Professor Hall calls them. Of these, the more distant, the prehistoric, come only slightly if at all to the surface; but insanity teems with reminiscences of a more recent stage—a stage which corresponds to the somewhat rapid transition from uncivilisation into polite society. Of all that history a review were well worthy of our consideration, a revision of the salient points in comparative physiology which denote the several developments of animal consciousness, as one function after another became for the moment the prime activity in the life of the species. However elaborate we may make our study of the histology of the present-day brain, we cannot by that means understand much of its significance, but only its mass and its complexity. It is only by recalling how, in previous forms, so far as we know, the consciousness embraced one function after another, and how each was superseded by another, but not entirely lost, that we can appreciate the multitude of important relations which lie buried, and not very deeply, beneath our present conscious level. And as we glance at a few of the most important phases of consciousness in its evolution, we should note all through a few general facts whose importance may become apparent later on. In particular, let us remember that character is stigmatised not only according to the things which the mind attends to, but also by its mode of regarding them. Two minds may embrace the same things in their experience, but their embrace of them may be entirely diverse. Insanity reveals an alteration from the normal consciousness in both respects. In insanity there is always under-attention to some things and over-attention to others. But the mode as well as the range of attention may be insane. Insanity is a failure of the normal personality, whereby the mind is reduced to a lower level of consciousness. And the modes of consciousness which it reveals are vestiges of a previous stage of our evolution. For example, perhaps the most important mode of insane consciousness, the fugitive, is one to which we shall often refer. It is a mode of mind which is afraid, has the sense of being hunted or persecuted, is suspicious, unfriendly, and querulous. That is obviously a reversion to a kind of mind which was normal to a stage of evolution in which life was much more dangerous and much less kindly than now. But it is merely a sample of many such vestiges.

Our study resolves itself into an account of the human mind as the physician ought to know it, whether in health or in disease. To him, mind is an evolution which has come by very slow and gradual stages, never making a sudden step. There is no point where we can say, Here is mind for the first time. For such a development, including, as it does, an organic as well as a spiritual evolution, we must presume, as Weismann has taught us fully to understand, that nervous tissue, capable of indefinite multiplication, carried with it from the first all the possibilities which it has since achieved; and that it is now potent for whatever it will achieve for all time. The modifications which it undergoes in each individual, and which are characteristic of the species, are not lost as evolution proceeds, but only become subimposed. All previous modifications are preserved—those which are vital as still active differentiations, but many of them as vestiges or rudiments. These functions necessary to organic well-being are developed in embryo and are mature long before the intelligent functions. Intelligence does not take note of them as subjective, does not embrace them within its experience in adult life, though we may conjecture that infant consciousness is aware of them. The embryo, or at least the infant, has perhaps sensibilities which become lost as others are acquired. In particular, it would seem likely that visceral sensibilities exist at an early stage and are lost when the special senses become actively engaged, just as we know infants have movements which they subsequently lose. Be that as it may, it is an obvious fact that, as character develops, the mind undergoes a great integration, carries forward only a few things in life and drops all the rest. And the important thing for our present purpose is to remember that, though nine-tenths of experience drop off the *mind* and leave no trace in it, it is not so with the brain-cortex. Facts drop into the brain, not off it. Consciousness in adult life is habitually fragmentary; even vivid experiences are attended to only for a time; people are aware of only one thing at a given moment. The mind, therefore, can only be understood as grasping one small bit after another of a vast range of possible things which are for the most part unconscious. Personality grows as the individual conceives of himself as the same person in a series of ever-increasing relations; it is strong according as some definite purpose dominates all experience; and cerebral differentiations corresponding to such integration must be presumed with special reference to inhibition. Personality then is as much an affair of brains as of the intelligence; and its disturbance in insanity is to be regarded as the exhaustion of those nervous mechanisms which represent purpose and purposive conduct and the consequent revival of irrelevant activities. These things must be borne in mind as we try to interpret the facts of biology, as well as the facts of experience.

Of the consciousness of the lower animals we can say very little that will be generally acceptable. For the beginning of organic reflexes we must go right down to the most primitive organisms, whose nervous function apparently is entirely alimentary. Their nerves respond only to food; their movements are concerned with the appropriation and with the disposal of it. At first there is no perception of food as distinct from rubbish, and we may question if even fairly advanced organisms perceive their surroundings. At such a level we can learn but little, except to remember that there was a time when animal consciousness, if it may be called so, was directly, particularly, and wholly visceral. And this visceral sensitiveness extended, we may suppose, to the respiratory organs when they appeared, to the cardiac, to the reproductive, and to the others in turn. Of such beings it seems mistaken to speak as if they had a personality, for that word implies a conception of oneself in relation to other things; and these creatures are probably not conscious of themselves, but only of parts of themselves. But we may speak of them as having a character—a visceral character. Right up the line to mammals the fact is in general apparent that the nervous system in certain cases, and presumably consciousness, have reference primarily to what is happening within the organism in all its parts—a particular subject-consciousness; and the first outward reference which the organisation seems to have is towards things which have an almost purely nutritional purpose. This preponderance of internal consciousness is carried up even into the big mammalia, is especially notable in some of the domesticated varieties, and in my belief characterises in considerable degree even some human individuals. It is probably an error in interpretation of a cow's movements, for example, or of a sheep's, or of a pig's, to imply any great perception of the environment. The history as well as the anatomy of the herbivora suggests that the feeling of the animal is still very largely visceral. The character of these animals can best be described as still nutritional, with the addition of more or less acute phases of sexuality. Attention to stimuli arriving by the special senses is but an interruption of a consciousness which is still chiefly determined by internal stimuli. And to such a mode of consciousness, with its characteristic over-attention to the bodily states, insane patients are often reduced. Indeed, we are familiar with it in the hypochondriac. But its most obvious revival is in visceral melancholia and in delusions of persecution which refer to bodily symptoms.

In relation to defence and offence, especially in the carnivora, the rare development of special sense—eyes, ears, and nose always on the *qui vive*—betokens a range and activity of object-consciousness, which of course has developed gradually, but which now for the first time is of the most obvious importance. It would be more true, for example, to speak of the character of the fox, the hyæna, the leopard, as predatory or fugitive, offensive or defensive, rather than as visceral. We may regard their consciousness as the converse of the visceral—a habitual attention to the environment, of which internal stimuli are an interruption. That preponderance of outward relation has of course persisted; and there are individuals in whom it is notable—persons who are uncomfortably aware of the smells, the sights, and the sounds of their neighbourhood; or who, without discomfort, are always noticing things. And of either kind of consciousness we have to note how little the attention really grasps of all the things which the brain carries

Every creature has its mode of consciousness, which largely determines the range of its attention, and all its other nervous processes are subconscious.

It is, however, in the social functions of animals—whether merely as a family, or in a flock, or school, or herd—that we begin to have a suggestion of an evolution suitable for the development of personality. Still capable of much visceral sensation, still wide awake to environment in general, the attention of animals in a family or flock has, for the first time, an opportunity of relating to systematic activities. Now becomes manifest the domestic consciousness, with all that that entails of attention to each other, and upon which the whole fabric of social feelings is based. In such conditions also arrives that kind of object-ward attention which is an advance upon the merely predatory or fugitive, which relates to the environment as amenable to work—the construction and care and fortification of the home, the colony, the city. In this is the beginning of the pre-eminently human faculty, one which, not content with the world as we find it, proceeds to a reconstruction of it. The prime character of early man—so that Carlyle distinguishes him as tool-using—is his constructive faculty. In the evolution of these activities which regard the world as a place to be made fit for human habitation is the beginning of all that phase of the mind which we may speak of as purposive. Organisation, providence, ambition, ideality, became possible only as men regarded the world as a place to be taken in hand—different from the point of view of the self-regarding beast, which takes things as it finds them, except when it destroys them. This constructive mode of consciousness is of course still characteristic of our race—so much so that every generation sees a new face upon the world, an evolution of the environment to which the individual must be educated. And, in insanity, when purposelike activities are no longer possible, the fashioning of demented tools, and futile constructiveness of various kinds, are quite common.

In social life also comes the opportunity for the thinking consciousness—a phase of mind which we cannot recognise in any considerable degree on a lower plane. The first form of it was probably very practical—the thinking out of means to secure very near ends. But only in the security of a society was there possibility also for the speculative form of thought—the kind of intelligence which, curious and filled with wonder, began to ask questions and to seek a reason for things. That kind of mind has probably become rapidly more common with the pacification of the world. It is to be noted as in a cycle which, leaving the more abstruse problems to the philosophic aristocracy, comes back again to the practical problems with which science deals.

But the important fact to note concerning early gregarious life is that thought and feeling and purpose were probably determined by what we may call the subordinate mode of consciousness—one which looked at things from a social and not from a strongly individual point of view. Each man and woman grew up to regard the tribe as the main thing, and the self as of subordinate importance. But primitive man fell away from this mode of consciousness, when he began to regard himself as more than all the rest. When, in a society made safe and comfortable by combined effort, man orientated sufficiently to regard himself as apart from his neighbours, personality as we understand it began. Departing from tradition, men here and there achieved the conception of a personal career; and in that conception, other men and women, as well as everything else in the world, were to be made use of. That notion of a career—from the most humble to the most proud and ambitious—is implied in the habitual human consciousness of to-day. And it is in the struggle between each other that the greatest stress falls upon the organism. When men learned this too great self-regard, insanity as we know it became imminent. For in the sense of individual responsibility and in the sense of personal failure are the greatest subjective occasions of mental disorder.

In polite society, however, no matter how occupied a mind may be with a personal career, good manners and a civilised morality have become essential. These are often lost in insanity, and the consciousness frankly and rudely disregards other people. Says Maudsley: "By insanity of mind is meant such derangement of the leading functions of thought, feeling, and will, together or separately, as disable the person from thinking the thoughts, feeling the feelings, and doing the duties of the social body in, for, and by which he lives. . . . However it has come about, whether by fate or fault, he is now so self-regarding a self as to be incapable of right regard to the not-self; altruism has been swallowed up in a morbid egoism." That denotes what, in more purely psychological terms, Bevan Lewis calls the "rise of subject-consciousness" in melancholia. For it is chiefly in melancholia that reduction occurs from the subordinate to the self-regarding consciousness. In other insanities, notably in moral imbeciles, it is obvious that the subordinate consciousness is still beyond the reach of many. And in perhaps the majority of men a domestic ideal, which still does not have great regard to the good of the race as a whole, is the utmost attained to. Yet is our evolution, if anything, a return, on a higher plane, to that primitive but very moral purpose which determines to think most of people in general and not of oneself in particular.

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But besides all that the human brain has acquired from its ancestral experience, we must take note also of its prospective developments. I have said that all possible evolution of human brain was inherent in nervous tissue from the first, and that the nervous system embodies in some degree not only all the unrecognised memories of previous experience, but also the potencies of all that awaits human character. Whatever men and women are going to be fifty, or a hundred, or a thousand years hence, is inherent in the brains, though not present in the minds, of to-day. For the unique quality of nervous structure, as compared with other kinds of tissue, is its plasticity, that it is apt for differentiations of infinite multiplicity appropriate to any and every condition of life. Men's minds grow and change in relation to an environment which is steadily becoming more complex, and in that evolution an unlimited plasticity of cerebral structure is implied. And while, in average mortals, new character only comes in response to altered conditions, the foremost of the race are those whose personality is precocious, whose minds and whose brains are in advance of their time. Here again we must suppose an automatic maturation of nerve-structure, a preparedness of mechanisms for conditions which have not yet arrived. And that precociousness of brain is not only characteristic of genius, but pertains in some degree also to all who are of robust cerebral vitality, and is pre-eminently the quality of the imaginative mind. As a quaint conceit we may suppose that some people are born into a world which does not recognise their genius, and which calls them insane, persons who perhaps know, or who at least surmise, a great deal too much. A great many quite intelligent mystics believe that we are on the eve of astounding spiritual developments, that the coming race, for example, will develop sensibilities which will transcend the material limitations to which we now submit. A considerable number of the insane suggest that they possess these occult powers. Apart from these, it is certainly the case that many patients, in contrast with the moral imbeciles, find their way into asylums as to a refuge from an ungentle world not civilised enough for their too self-sacrificing natures.

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These then are the modes of consciousness to which the physician must have regard if he would understand either acute insanity or the much more common and not less important cases which will be described presently under the term, abnormal mental variations—cases of abnormal cerebral development manifest in all forms of eccentricity. They are modes of consciousness which have been arbitrarily emphasised, and we might elaborate upon them indefinitely. I have selected them not only because they seem to designate well-marked biological stages, but also because they become manifest in types of insanity. Melancholia, for example, is a relapse to an almost exclusive self-regard, a loss of outward relations. Mania is the converse—an excess of objective attention, either as exaggerated and incoherent notice of what is in the neighbourhood, or as extravagant outward activity, and generally both. Dementia, if anything worth calling consciousness survives, probably denotes a relapse to the visceral phase, which is also conspicuous in some melancholias. Delusion is an unrelated survival of primitive thinking. And in insanity the less systematic modes of mind also hold the field. Nothing is commoner in our asylums, for example, than the fugitive idea; a bald and unblushing devotion to the domestic ideal of primitive man is common enough; predatory instincts are laid bare in many chronic insanities; sensory hyperæsthesia is much more common than most physicians are aware, and is probably very acute in many neurasthenic melancholics, and in delusional and hysterical patients; daft constructiveness, outvying even the futility of a benighted beaver or ant, constitutes the entertainment of many a happy dement; and less advanced cases spend long hours in inept imaginings. With less assurance we may suppose that some of the over-activities of our patients are modes of thinking and of feeling appropriate perhaps to a stage of evolution still a long way hence. And these modes of attention are still more evident in the abnormal, still short of certifiably insane. Any observant physician will be aware of a large number of acquaintances whose minds are abnormally visceral, or predatory, or fugitive, or constructive, domestic, self-regarding, given over to futile observation, or lost in inept speculation. For the mind of man has not altered very much in kind, but only moves on a different plane, and has to concern itself with new kinds of things.

(2) Personality and Subconsciousness.—In attempting to conceive the cerebral relation which mental processes admittedly have, the physician should be guided by his familiar knowledge of the interdependence between brain and mind on the level of simple functions. We all habitually think of vision or of hearing, for example, of articulation or of penmanship, as dependent upon cerebral activities on the Rolandic level. Yet these are, or may be, mental processes. And what is true of them is true of the higher functions. Integrity of the præ-frontal cortex is essential to proper feeling, to sound thinking, to right conduct. It is equally futile to explain the mind as independent of cerebral processes, and to explain it as wholly determined by them. The relation is one of many of an analogous kind, much more subtle than that of simple cause and effect. But a great deal of unnecessary mystification has been made of it. Not that there is no mystery. But there is certainly no greater difficulty than baffles us when we attempt an account of other

ultimate processes. An erudite account of a telephone system, for example, really explains nothing satisfactorily. In the end no one understands the relation of the voice to the wire; and that is the kind of mystery which surrounds the relation of mind to brain, and the only one which is at present quite beyond our horizon. For the cerebral function has to do with forms of energy and nothing more. Brain is simply the vehicle of words and of other things heard or seen or otherwise perceived—energy which travels hither and thither in the cortex, to reappear in conduct. And to appreciate this relation of human energy the physician might do worse than think out this analogy of the relation of life to the telephone. For the telephone and the phonograph do just what the brain does, though by different means. Especially when we learn that the telephone and the phonograph have been combined—adding a function of retention to that of conduction—is there aptness in the simile between the electrical and the cerebral mechanism. Both instruments, for example, depend for their function upon something which we may call tone—electrical in one case, nutritional in the other. If the cortex is not nourished it will not conduct properly; but in health the nutritive processes maintain the lines in good order for the passage along them of the appropriate currents. Further, both instruments depend for their efficiency upon correct and intact connections. Every little while interruptions occur because of wrong contacts, and the energy is diffused in wrong directions. One cannot insist too often that all the positive signs of insanity are of this nature. We have been too slow to appreciate the lessons to be derived from maladies of the sensory and of the motor areas. These we have long ago learned to consider as manifesting paralysis—loss of function; and diffusion—disorderly function. Excitement, delusion, fear, and other positive symptoms of insanity are but incoördinate excesses following upon a paralysis of those mechanisms which normally command the cortex. There is no difference in the kind of effects which disease has in insanity and those which it has in tabes, in multiple sclerosis, or in a lesion which affects Broca's convolution. The difference is only one of situation. In each case the defect is that normal connections have failed and movement passes by wrong channels.

We must, however, take note of a feature of nervous tissue which is essential to subconsciousness. Next to its conductivity, which is its first and obvious function, the brain is an organ of retention. And retentiveness depends upon the nutrition of differentiated structure, just as every other function does. We are very liable, if we think of our brains at all, to conceive only of that function which is engaging our attention for the moment, or of our habitual activities. But, if memory has any organic significance, it implies that every experience to which we attend—and others as well—leads to a characteristic differentiation; and that the mechanism of it is maintained indefinitely and is functionally active to some extent all the time. To furnish an image of something that we saw or heard an hour ago, a year ago, twenty years ago, the brain must have preserved an activity in a definite and appropriate set of nerve-cells and fibres. One must therefore conceive of the mind as afloat upon a vast sea of movements, of which only a small part comes now and again into relation with it and engages the attention. Here again we may revert to the analogy of the telephone, and remember what an ocean of words is streaming all day long through its countless wires. Sometimes, just before the contact is made for you which isolates the message that you want to hear, a hum as of a million voices vibrates in the instrument until suddenly the proper voice is focussed. In the cortex a similar relation obtains. From infancy onwards, personal experience has initiated a million activities which persist indefinitely in the brain. Moreover, other cerebral activities which are independent of individual experience occur automatically—instinctive processes which are the inheritance of the race. Axioms of life, for example, self-evident propositions which the mind accepts as facts the moment they are presented to it, imply a cortical structure which was active before the occasion for its use arose, just as the act of sucking implies an automatic maturation of a motor mechanism. But both these activities which experience initiates and those which are instinctive lie buried, for the most part, beneath our consciousness. They come within the attention only as occasional facts, when contact is made with them in the course of a train of feelings or of ideas, or when an outward stimulus occasions our notice of them. Subconsciousness is as vast and as real as are the unheard, because disconnected, messages of a telephone system.

We may compare this development and differentiation of cerebral mechanisms to what occurs in the growth and development of bone. Nerve-cells and fibres maturate and become differentiated very much as bone cells do. There are to begin with what we may call centres of cerebration—foci of developmental activity, which are common to the race, and whose diverse proportions determine individual variations. For example, the centres of vision, of hearing, of sexual feeling, maturate automatically at different times, and character is modified according as one or other of these (amongst a host of others) is strong. But structure is determined also, as in the skeleton, by functional factors. What the mind attends to, that will the brain become apt for, as surely as stress moulds bone.

The relation of the mind to subconsciousness has been made the subject of much unnecessary mystery. We need achieve no new conception in order to understand it. If we analyse our mental processes we shall discover that attention to an idea or to an emotion is not different in kind from a simple sensation. If a thorn pricks, we are aware of the prick simply as such without thought of the rest of our body. And attention to a mental fact is equally simple. We attend to a fancy which jumps into our mind, or to a memory, long hidden, which something at hand suggests—both of them derived from subconsciousness—just as we attend to a prick. The difference is that we do not locate the object of our attention in the former case. We have not developed a mechanism for the location of mental processes. As I have suggested, consciousness has spread in its development as each system after another became the subject of nervous function. When at last nearly every part was possible of discernment, the mind was in a position to perceive the body as a whole. But there is nothing different in the perception of one's own body and the perception of any other. That kind of self-consciousness then—the bodily self-consciousness—is nothing strange. Nor is it strange that we should be aware of nerve movement which is occurring in our brain any more than of one which is in the big toe. The only difference is that we do not locate the former. The nervous activity which underlies an idea simply does not appear to us as occurring in any particular place. Hence of course the difficulty of realising that mental facts have physical facts to correspond. But the brain is one of many viscera whose nervous activities we do not locate. (No healthy man locates his pancreas.) None the less of course the experience, the content, which accompanies cerebral activities, is as real as that which we recognise as having to do with a toe or a finger. And, as the mass of these unlocated cerebral facts multiplied, men and women—or at least some of them—have come to regard their spiritual selves as themselves and their bodies as secondary. Character or personality has evolved ever since that time. With the spiritual aspect of such an evolution we have nothing now to do. Our business is to remember that every tradition appropriate to the race has had a nervous structure to correspond with it in the brain of each individual who accepted it; and so also with every personal experience. At school, in the family, at college, in the workshop, traditional discipline is constantly at work, and but for the force of individuality, moulds all men and women to something very near a given pattern. And as men think, they choose as a rule the kind of life which their training has suggested, with just a slight modification due to the personal equation, so strengthening by act of will the force of education. All that process simply implies a very definite and very stereotyped kind of nervous differentiation. It implies that in each individual the great mass of cerebral activities hardly come into contact with personality at all; and, conversely, that those activities which the man selects as appropriate to himself become strongly integrated. A man's brain is commanded by the functions to which he devotes his attention. And such a stability is to be expressed physiologically in terms of inhibition. Inhibition is a nervous process which we all recognise in what we may call its focal form in such mechanisms as control cardiac, respiratory, cystic, and rectal movements. In these an isolated and recognisable group of cells and fibres control the activities within their physiological sphere of influence. But the process is one which pervades the whole central nervous system. Indeed we cannot understand cortical development and integration at all except as a serial evolution of one function after another, each having a sphere of influence in which subordinate mechanisms are under its control. And in the mechanisms of the stable mind this process is one in which every cerebral activity which comes within the province of the intelligence is under the inhibition of the centres which represent personality.

We are now, then, in a position to state less vaguely the relation of personality to subconsciousness—a conception without which, or some substitute for it, any general understanding of sane or insane life is impossible.

Subconsciousness is that whole mass of cerebral activity to which the mind does not attend. At any given moment that is nearly everything that is occurring in the brain. By means of attention the mind handles, so to speak, one of the brain threads at a time, or several in very quick succession, does something with each, and drops it again. Some parts of subconsciousness never come within attention at all; some come once into it and never again; some are very frequently there. Further, the mass and kind of subconsciousness varies with different constitutions. And it varies, and the mind's relation to it varies, with individual experience and habit. The essential point is, however, that at the moment of contact, when the mind is conscious of a cerebral activity (not *locating* it), something happens. As the mind moves along a train of thought, for example, the thread of cerebral activity which it pursues is not left the same as before. Next time it comes into consciousness it is different. The irrelevant and casual things to which we attend we need not now consider, except to say that their subconscious existence explains, for example, a vast deal of the sayings and doings of the delirious and of the insane, who fetch forth from their recesses many strange and hidden and sometimes horrid things with which their friends never knew them to be acquainted. But a great deal of life is consecutive and coherent and becomes woven into the personality. To become concrete, take the commonest and the average personality whose central feeling and idea and purpose concern themselves with livelihood. At the foundation of that there is the domestic consciousness—wife and children, the hearth, bread and butter. For that there is certainly a cerebral mechanism—a centre diffuse in form, but definite in structure. We have not located it yet, but surmise that it is in the præ-frontal region—the area for the images of acts; for personality is determined by its expression of itself—what a man conceives of himself as doing and saying. All consecutive experience then comes into relation with the man's notion of livelihood. From religion to silly pleasures the criterion of their effect upon his position and comfort and prospects is the constant one by which the man gauges things. And that means a great integration as well as a great spread of mechanisms. The process is one which occurs to some degree in every mind, for even the most versatile have one or a very few notions to which they most attend. All through life, then, one activity after another is coming into relation with these highest and most central mechanisms. Each point of contact modifies both. On the one hand, the primary mechanism is modified, extending its fibres so as to meet and, if all goes well, bring that other within its sphere of influence. On the other hand, the secondary mechanism is modified conversely, just to the extent to which it has been subordinated to the primary centre. A sane and successful life is one in which every conscious experience enlarges the range and adds to the power of the organs of personality. The occasions of insanity, over and above the organic causes which interfere with nutrition, are those experiences which are in conflict with the main purpose of a man's life. To revert, for example, to the average personality mostly concerned with the hearth, the commonest occasions of insanity are domestic affliction, or domestic dispeace, or financial troubles, or social disgrace. It is friction which breaks a man's spirit—when the circumstances of his life thwart his purposes, or when impulses bred of vicious organisation or wayward habits pull against them. His brain is an arena of conflict between activities which he has made his own and others which are being forced upon him. Then, if personality is not strong enough and purpose fails, the attention becomes engaged with the irrelevant and perhaps the insane things of a subconsciousness that is without rational direction or supreme government.

II. Index of Mental Functions and Symptoms of their Disorder

It is a very doubtful question if psychology can ever become very scientific, and if the knowledge of the physiology of mental processes can ever be exact. The chief difficulty in the way is that analysis is futile. The solidarity of the mind is too great, the brain is too compact, to admit of dissection. Hence all attempts to describe the faculties of the human mind are misleading, and even descriptions of particular processes are rarely convincing. But some attempt must be made to indicate the most important aspects of mind, or, as physicians should regard them, the mental functions of the brain. Even if our description of them fails of reality, it is still essential to note certain kinds of processes which one should habitually observe.

Instincts.—An instinct is an automatic activity, the common inheritance of the race, made manifest in appropriate movements, and, in its higher forms, realised in appropriate feelings. Instincts for vital movements are familiar. One need not enumerate them. But comparable to them, at least in their physiological aspect, are other less elementary instincts which subserve a social function.

These also betoken an innate development which, in every normal brain, is more or less mature before the occasion for their manifestation arises, although, as always, practice makes them more perfect. An enumeration of these must needs be arbitrary and incomplete; but we may note those of which we most commonly have to observe the failure. The instinct to prolong one's own life and to guard against danger comes first. An arrest of its development constitutes the predisposition to suicide, which is often hereditary; and homicidal tendencies mark those who fail of the instinct to respect the health and life of others. Pain and discomfort are generally the occasion for their active manifestation. The normal man is so constituted that pain and discomfort, or the knowledge of them, prompt him to alleviate them. Mutilation of oneself, starvation, and the many degrees of disregard of pain and of discomfort, all very common in insanity, betoken an impairment of the life-preserving instincts. The mating instinct is another, whose arrest, or excess, or perversion so commonly appear as signs of cerebral disorder. Next in importance, perhaps, comes a group of instincts which we may call the domestic—instincts which have developed in relation to family life. Whatever happens, the instinct still persists which attaches us to our own folk; and few symptoms of mental disorder are so tragic as that perversion of the strongest of the domestic instincts, the maternal, which culminates in infanticide. Going beyond the hearth, the step is easy to that instinct which prompts men to get on in the world, and to that other which enjoins upon us to seek the company of our fellows, and to earn their favour. Departures from these, or excesses of them, are commonly to be noted in disordered minds. And lastly among the social instincts we must take note of a patriotic instinct, the highest perhaps of all, for, although we may conceive a cosmopolitan ideal, we can scarcely be said to possess a cosmopolitan instinct. The care for our country and for our race is certainly inbred; and, though we do not often have occasion to note a failure of it as a symptom of actual insanity, it is interesting to observe in how many unexpected quarters recent events have made evident the defect of it. There still remains the religious instinct, the instinct to reverence and to worship, which, whatever we may think of religions, is the subjective basis of them. That instinct, as every one knows, often finds abnormal expression in disease or falls into abeyance.

It were easy to multiply the instincts and to refine upon them, but those enumerated are sufficient to suggest the others. They constitute the foundations of well-organised brains. Any lack of them, any perversion of them, any extravagance of them, betokens disorder; and in gauging his patients' mental health the physician must take careful note of them. And let me repeat for the last time that we must conceive of a development of mechanisms to correspond to all of them,—a maturation of cells and fibres which represent them,—and a subconscious, occasionally conscious, activity in these instinctive centres. These are the primary points of cerebration, so to speak, whose automatic activity determines personality.

Interests and Tastes can be more summarily dismissed, for the reason that they are more varied and less universal. But it will be obvious to any one who has watched the development of children, or who has thought of it, that innate cerebral organisation determines to a large extent the preferences of the individual. There can be no doubt, for example, of a specialised æsthetic taste in some cases, of an innate greed for rich colour or fine form or good sound. A useful observation has distinguished between the ear-minded and the eye-minded —those whose brains have the avenues of hearing or of vision specially active; and, more commonly, we have to observe practical tastes—an interest in making music, or in producing colours and forms, and various other outward activities, of which the love of sports is perhaps the most usual. In a well-balanced mind æsthetic interests are well blended, but the normal development is always towards practical pursuits. Short of the gross defects of vision and of hearing, and the gross motor failures with which we are all familiar, slighter degrees of defective interests are often to be noted as characterising bad brains; and, in insanity, inquiry always elicits an account of a change in the patient's pursuits.

Humour, Temperament, and Disposition.—These constitute still another kind of innate cerebral characteristics. The day of temperaments is past. But we have still to note that many people fail of that good humour with things in general which is the good fortune of a healthy brain, and that, in active disease, an extravagant optimism is common. So far as is known, good humour is determined by a right proportion of expenditure and revenue of energy. It is when the brain has enough vitality and to spare for all the calls made upon it, and—what is apt to be forgotten—when it is allowed to expend its surplus, that good humour comes. And temperament is determined by the prevailing humour. The term disposition merely indicates the expression of temperament or of humour. It signifies general feeling made manifest in conduct—*bonhomie*, kindliness, tolerance, or surliness, sourness, moroseness, hopelessness, as expressed in converse with our fellows. In all disordered states of brain the disposition is changed, though often pleasantly as in the early stages of mania.

SENSE-PERCEPTION.—The functions to which allusion has been made are functions in which the innate constitution of the brain is obviously of the first importance—functions which depend upon the original vitality of its parts. And it is still true of those which we are about to consider, that development, up to a certain point, precedes experience. But experience now plays a more obvious part. If the eye is not filled with seeing or the ear with hearing, it is still true that sense-perception is a function or group of functions which depends very largely upon experience for its development. And these functions, which we are too apt to regard in their primary aspect—as merely subserving sense impressions—furnish very largely the data of intelligence. We think and feel and act after we have seen, heard, tasted, smelled, and handled. We must, therefore, conceive of each sensory tract not merely as developed in relation to the objects to which it obviously refers but as woven into all the mechanisms of the intelligence. To conceive the nervous basis of mind, we must think of an extension into the mental areas of these mechanisms which we commonly associate with acts of simple observation. The visual system, the auditory system, and all the other sensory systems, are essential elements in the framework of intelligence. And in that conception we must include the visceral tracts whose activities in health do not obtrude themselves upon the attention. The ovary, the heart, the intestines, are intimately related sensorily with the mechanisms of feeling, and constitute a large area of subconsciousness. Disorders in all these realms are familiar—paræsthesias, illusions, hallucinations. Sometimes the disordered activity is high up among the mental mechanisms, sometimes in what is usually termed the sensory area, and sometimes peripheral. It is not always easy to discover the extent of defective sensation in mental cases, but there are few in which, if the physician seeks for it, he will not find some disturbance of function. (It may be noted here in passing that there are equally intimate and elaborate relations between the mental areas and the motor tracts. Activities in these connections, however, are chiefly outward, and disorder is more likely to spread from mind towards muscles than contrariwise.)

MEMORY.—One of the prime characteristics of brain as an organ of intelligence is the fact that it retains some kind of image of the impressions which it receives. This function of retention, though most obvious in memory, is implied in all mental processes; or, in other words, memory comes into every act of the mind. As has been said, attentive observation does something to strengthen, that is, to make more facile, the images which the brain stores; for attention generally implies that we are associating the impression of the moment with some train of thought or feeling which is more or less habitual. But we do not know that attention does more than that. Probably all impressions which reach the brain—and they are innumerable—have their characteristic effect; but, if not associated with personality, go to swell the mass of subconsciousness. That this is so, is supported by the facts of delirium, in which the brain reproduces innumerable things heard and seen which had lain quite without the voluntary recollection of the patient. It is indeed doubtful if the brain evolves much material for conceptions, though the process is not incomprehensible. More likely the new combinations which occur, for example, in dreams and in active imagination—new scenery, new incident, new personality—are chiefly compounded of images fished up from subconsciousness and strung together. But the products of retention are more orderly associated in rational memory. And here we must observe a fact which comes second only to retention in its importance to character—that each stimulation of mechanism (short of fatigue) increases its facility. Practice makes perfect; familiarity deepens impressions; repetition strengthens memory. As I have said, any retained image which can be recalled probably implies a mechanism in which there is constant, though subconscious, activity. But, perhaps for want of renewed impulse, these currents, it may be, lessen and cease; while those habitually augmented by restimulation are perpetuated. And the mechanisms can be no other than the same mechanisms primarily concerned in perception, or perhaps an extension of them. To learn a fact and to recall it imply very comparable processes.

Different degrees of association and of repetition characterise the distinction, useful to the physician, between old facts, recent facts, and permanent facts. In investigating a patient's memory we try to discover how far he can recall the facts of yesterday, or the facts of ten years ago, or again a fact which has been all along the same—as, for example, the Queen's name, the date of Waterloo, or where the baby Moses was found. In all mental disorders there is some impairment of this function, and in a few, especially in alcoholic and in delirious insanities, the defect is quite characteristic. It is chiefly in disease that we discover also the importance of the observation of three phases of memory—retention, recollection, and recognition. Defects of the first of these alone are practically unrecognisable. But it is very easy to detect defects of recollection, as, for example, in a patient who observes and clearly apprehends a fact, yet five minutes later cannot recall it. And of recognition—the function whereby we recognise the recollection as the thing we have been seeking for—disorders are common, and most obvious in cases characterised by mistaken sense of identities. A very interesting phenomenon, most familiar in the senile, is an extravagance of recollection which we may call reminiscence. The patient lives and moves in scenes of fifty years ago, the attention dwelling upon images which may have lain subconscious for half a lifetime; and, in minor degree, the process is common in many disordered brains. Lastly, we have to note a very difficult function—prospective memory, the ability to remember forwards. This function is one closely related to purpose and intention, and is very commonly, and often early, disturbed in mental disorders. The inability of the insane to carry forward their plans is often more notable than their disability in conceiving them.

THOUGHT, INTELLIGENCE, COMMON SENSE, BELIEFS.—The fact that so few people can really be said to think, taken with the average amount of level-headedness and sagacity, and the average amount of proper conduct, speaks volumes for the subconscious activities of brain. In the rational sphere common sense is to knowledge what the domestic instincts are to the affections. Most of the sagacity of the average person is innate or intuitive knowledge, and not the fruit of logic. This implies then an abundant activity in nerve mechanisms which automatically determine opinions and beliefs. Thought indeed, in its elaborate modes, does not often call for investigation in mental disorder. It is a delicate process which, in those who have practised it, disappears very early in disease. In its place we have to note, on the one hand, premature inferences, and, on the other, extravagant imaginings, both alike due to an abeyance of corrective judgment. The foolish and the deluded seem to accept blindly almost any opinion that their brain serves up to them; and that this disorder is a product of subconsciousness is testified to by the fact that these people invariably prefer their own explanation of things rather than any, however plausible, suggested by an outsider. The physician, if he tries, will find it hard, and hardest in the most deluded, to substitute one insane belief for another in the mind of his patient; those mentally disordered are notoriously not amenable to suggestion. Delusions then are generally intuitive products, although circumstantial facts may later be adduced to support them, and are similar in kind to the imperative ideas which we call obsessions. In obsession, the brain presents persistently to the mind an idea, often quite irrelevant to the rest of the patient's life, but eventually touching his personality. These phenomena partly indicate a disordered activity from which some imaginative opinion rises into consciousness and dominates it; but they betoken diminished rather than excessive thinking. It is the function of corrective judgment, the ability to weigh the facts carefully before coming to a conclusion, which is at fault. Gullibility is a very common feature in persons who are of the insane diathesis; and a sense of probability is conspicuously lacking in the insane. These defects obviously betoken a want, or an impairment of common-sense. Short of delusion, the practitioner will often observe minor failures of this kind—facility, opinionativeness, extravagant expectation—due to an exhaustion of judgment, and likely to be followed, if not corrected, by positive delusion. Quite as often also—and this is important—we observe in the unhealthy mind persistent confusion (see later under Confusional Insanity). In one sense confusion is normal to all thoughtful minds; it characterises our state in regard to every question concerning which our minds are not made up, and the more thoughtful we are the less sure we are. But confusion in the insane very often refers to things which, in the sane mind, admit of no question. It is essential to mental stability that we should take a great deal for granted, that we should assume that what everybody believes is true, that we should continue to hold beliefs which we have proved. Any of us would become painfully confused did we, as these people do, entertain serious doubt as to whether this is really Wednesday, whether it is wise to put on one's boots, whether food is healthy or poisonous, whether we can or cannot do without breathing. Such are the failures of common-sense familiar in mental disorder, and this perplexity, in a minor degree, characterises many slight affections. But some observers make the mistake of assuming that patients are confused because they talk confusion. Deluded persons are not really confounded; on the contrary, foolish assurance is much more obvious in the insane than confusion is. Doubt is characteristic of less grave disorder.

Feeling, Emotions, Affections.—The term feeling is best confined to the simple states of pleasure and of pain. It attaches normally, as we know, to certain kinds of experience, to certain ideas, and sentiments, and emotions. As has been said, the absence of distress or discomfort betokens a nervous activity for which the brain has sufficient energy. So soon as fatigue occurs, whether from over-exercise of the brain, or because it is ill-nourished, pain arrives, and the patient is depressed. Then even the experiences which can commonly be counted on to give pleasure fail of that effect. Bad spirits, a defective capacity for enjoyment, are amongst the commonest signs of mental disorder, and call for treatment which will promote regeneration. In some cases fatigue induces the patient to dwell upon his own misery, and melancholia supervenes; or, if self-control wholly fails, he takes to extravagant conduct and becomes exalted and maniacal. In many cases, notably in general paralysis, there is no period of depression of greater amount than is common in sane persons. The joy which supervenes in mania and in general paralysis is to be attributed to the abeyance of fatigued mechanisms and to the activity of the less wonted centres—those which subserve imagination, and the sensory and the motor areas.

Feeling attaches to all emotion and in characteristic tone. Speaking roughly, emotions which add to the possibilities of life are pleasurable, and those which take from them are painful. Hope and fear are types. Physiologically considered, hope opens out avenues of activity; fear closes them. And these states are instinctive, and, in some degree, universal. We cannot fail to note, however, great differences in individuals—that some are instinctively prone to hopeful and others to fearful emotion. And we observe also changes in individuals which betoken impending or actual disorder. Extravagance in either direction is bad, but the unhealthy are, as a rule, fearful.

Affections are cultivated instincts—instincts for people and for things which have become elaborated by contact with them. And here again we must note differences in individuals as well as changes in the individual. Here also the observation is roughly sound that pleasure betokens a healthy mind. In the more sane, likes exceed dislikes, hatreds are few and friendliness common, sympathy should outweigh antipathy, tolerance exceed criticism. In the unhealthy, the balance is the other way, and in mental disorder the affections are gravely assailed. Suspiciousness, antipathy, loathing, anger, supersede even what were strong affections. And, on the other hand, persons not normally affectionate may, when intoxicated with their insanity, fall in love with all and sundry, see nothing amiss, dote upon objects almost vile.

Habit, Impulse.—On the outward side of mind, in the realm of conduct which, as has been said, largely determines personality, habit must be our first consideration. Its persistence is determined by that fact about nerves which we noted under the subject of memory, that stimulation of mechanisms increases, up to a certain point, their facility. What a man's habits will be is obviously determined to a large extent by his instincts and tastes, but not less by his experience and especially by his education. In a sane and well-ordered life habits are part of a symmetrical whole, fit with general conceptions, and do not interrupt or impede the even tenor of a man's way. Vicious habits, on the other hand, are rebellious and out of keeping, fail of the man's general approval, and so engender pain. Even in the well-behaved, disorderly or unrelated habits may be a source of much worry; and there is no commoner source of mental mischief than the constant knowledge of bits of life that are in discord with, perhaps directly subversive of, the whole. It is a truism to remark how easily habits are formed, and here we have to note the converse of what has been said about instincts. While it is true that subconsciousness makes great contributions to mind, it is equally true that a great deal of our personality sinks into subconsciousness. Almost as soon as we have thoroughly learned anything we cease to be vividly conscious of it, though we can perhaps at will make contact with it again. And this process of habit implies that vast numbers of mechanisms, whose organisation came into existence under our direction, fall into a place in subconsciousness in relation to all the others. In sanity the vitality of those subconscious mechanisms which we have chosen exceeds that of others. In disorder the hierarchy is disturbed by the excesses of centres not closely associated with conscious purpose. A man's brain, then, is strengthened by all habits which are consciously related to his general purpose and approved of. We all know, and there is no need to elaborate upon it, what kind of habits make for mental health. We take it as roughly true that average habits are normal, and are justified in scrutinising the irregular. Far more useful, however, than a comparison of one person's habits with those of another is the observation of change in the individual. And it is a curious fact that mental disorder is generally associated with a change from the correct towards the disreputable. It is not often that one is alarmed by an access of propriety in a patient. But any change, especially in adult life, is noteworthy. In the insane it is remarkable how great is the departure from the wonted, and that often without any apparent conscious impulse or intention.

Regarding insanity, as we must, as a subversion of self-direction and the ascendancy of disordered nerves, impulse becomes very important. But the facts regarding impulse are so obvious that we need not dwell upon them. It is enough to revert to the possibilities that lie under the surface of the human brain. In any of the mechanisms to which we have referred—mechanisms which in sane life are subordinated to a general purpose—an excess of activity, occasioned by illness and the failure of the normal, may spring an impulse upon the unwary mind. And such impulses are as strong as those which are occasioned by normal conditions and which earn the mind's approval. And so, in various insanities, but especially in alcoholism, and in the insanities of the degenerate, an impulse, however preposterous or dangerous, may possess the consciousness just as an obsession may swamp all thought; and, unless prevented, the impulse will be realised in act. Like obsessions, these impulses are to be discerned as they are more or less at variance with the patient's habitual personality. Homicidal impulse, for example, may as a rule be taken to be due to an activity in mechanisms with which the patient has not been much in contact. Less insane impulses are of course commoner, and, equally of course, less observed—impulses to do just not quite what would have been done in health.

Moral Sense and Sense of Fitness.—By instinct, by education, and sometimes by conviction, our minds learn to know and to approve the right. The difficulties that beset any inquiry into the nature of that knowledge are great. But, as physicians, we have to regard moral life as of the same nature as other activities of the mind. Whatever be the code to which we have been born, and in which we have been brought up, our apprehension of it implies the development of an organ to represent it. These mechanisms are no more, but rather less, detached than others; they are, like others, related to all conscious life, capable of contact with the great mass of subconscious processes, and subject to disorder and degeneration. A less dignified function, but strictly analogous, is that which approves what is fit and proper — a function which relates to manners rather than to morals. That our appreciation of the good and of the proper is largely instinctive there can be no doubt. The brain is prepared to react to moral as to other stimuli. Many patients manifest an instinctive disability in this regard, are not easily made to understand what is right, and, when they do understand it, are not convinced. And, just as we have perversions of other instincts, so also of this; and men and women are to be found who are carried away by a zeal for this or that other virtue which the level saint regards as one amongst others. So too, in mental disorder, the moral sense often fails and shamelessness supervenes; or an extravagant conscience condemns fiercely even the most trifling offences. Similarly, the sense of what is fit is so commonly lost in mental illness, that we hardly recognise it as a fact that the insane and the unhealthy-minded are unmannerly; and, on the other hand, we have sometimes to note an excessive punctiliousness as a symptom occasioned by cerebral change.

Purpose, Intention, Ambition, Ideals.—We have already considered purpose in considering personality, and need only remark here that the function of prospecting life and of choosing a line is to be discriminated from that which gives effect to what has been chosen. In mental disorders they may be separately affected. A patient may conceive of life in disorderly fashion, may think the wrong things desirable, and may choose them, yet may carry out his wrong purposes with healthy vigour. Or ideals may remain intact, yet conduct wholly fail to realise them—a disability which is too obvious and too familiar to require illustration.

Conduct.—Downwards and outwards towards the muscles all mental activity eventually trends, at least in the healthy mind. Thoughts are to be spoken, affections to be demonstrated, projects to be realised. And this process of expression of one's personality would appear to be the last and highest function of life, the final task imposed upon nervous organisation. That failures occur in this function is very obvious; in fact nothing else in insanity comes under our notice. It is as the patient looks, and speaks, and acts, that we judge of his sanity or of his disorder. As a rule we infer that a foolish remark implies a foolish idea in the consciousness of the patient, but such an inference is not always just. Just as, in thoughtlessness, we say and do what has no significance to us, so, in mental disorders, much of conduct is without consciousness. In all excited states things are said and done which the patient probably does not choose to say and do. The mechanisms of speech and of deeds have the bit in their teeth and are entirely undirected from above. Or, what is equally common, there is simple abeyance of expression. In mild disorders nothing is commoner than for the patient to complain that, while he can quietly and properly prospect what he should and will do, he finds to his dismay that he cannot do it. This is in particular the symptom of stupor, which is essentially a depression in the functions of expression. Sometimes stuporose patients remember what they have come through, and will tell you that they were labouring to say and do things, and failed. The contacts between the mechanisms of purpose and of conduct had not been made, or, if they were made, the tracts of conduct would not transmit currents. Lastly, we have to note that most interesting phenomenon, resistiveness, in which the nerves, not content only with not doing what is wanted, insist upon doing the opposite. From the experience of the golfer, or other artist, who finds his performance so out with his conceit of it, to that degree of resistiveness in which the patient becomes rigid at the merest suggestion of movement, all degrees of resistiveness are to be observed in mental life.

The Self in Insanity.—In sane life men control themselves, which is to say that they keep those parts of their brain in subjection which they prefer should be so. Insanity is the reverse, it is the subjection of the mind to a disordered brain. That is why it is fitting that, for purposes of insanity, one should regard mind in its organic aspect, so as to understand and be prepared for cerebral effects. It is true beyond any doubt that cerebral occasions mental activity; but it is no less true that mind influences nerves. It is quite obvious that nearly the whole brain can be brought into subjection to purpose and intention. We are then face to face with the problem of what becomes of the self when mental disorder has usurped it. And I have no desire to contribute to metaphysical futilities on this question. But if this relation of mind to brain is a fact, it has great importance in mental disorders. And I would ask the physician to revert again to the analogy of those incoördinations—such as aphasia and ataxia—which we know to be due to motor lesions. In such a case it may be the patient is aware that when he intends one thing his nerves and muscles bring about another. So in insanity there are no doubt cases in which the patient realises that these words and acts are not his. In most instances, perhaps fortunately, insanity is as sleep, or as a dream, and the patient remembers not at all, or but vaguely, what has happened. But in all cases of convalescence the mind clearly resumes authority, and in many cases, it may be, it has been conscious of its overthrow, and not without hope of restoration. To build upon that, or to try to explain it, is to embark upon sheer mysticism. Yet the physician who prefers to be a mystic will not fail of reward if, while he does all he can to restore the disordered brain, he thinks kindly of the sad soul who is tethered outside of it, and if, when occasion offers, he tries to reach it and to reinstate it.

III. The Insane Diathesis

I have remarked, and repeated, that the brain has in it the potency of all that human character is going to be, and that it is unique in its plasticity. It is the tissue which, above all others, must be prepared to take on new developments. Each generation must be innately prepared for new ideas and new habits. And such a condition implies a possibility of great fluctuation or variability in the individual. Physiologists sometimes forget that, and omit from their account of brains the facts of the personal equation. For an organ which is still plastic, which is still evolving, is sure to manifest great departures from the type. And, though we cannot discern in every case individual peculiarities of cortical structure, we certainly can observe mental differences. To take an exaggerated instance, nationality makes all the difference. For that reason, I think, we attach far too much importance to descriptions of mental disorders written by foreigners. Not that their observations are unsound, for in many respects they have taught us scientific methods in insanity. But the mind of a Parisian is not the mind of an Aberdonian; a Tyrolese peasant does not think and feel like a Yorkshireman, nor a Dutch cheese-grower like a cockney cabby. And both national and local and personal characters greatly modify types of mental disease. Hysteria in this country is not the gaudy type which it is in France; hystero-epilepsy is a comparatively insignificant thing with us; the mental facts of general paralysis and of alcoholism vary so much as to be hardly recognisable among different peoples; and (perhaps the best case in point) progressive systematised insanity of persecution, as described by Magnan, can hardly be said to occur in Scotland. Even in any one community the forms of mental disease are as important in their discrepancies as in their constant features. No two minds are alike either in health or in disease, and a great many minds are remarkably divergent from the average. And, as we know is generally the case with extreme variations, these odd minds are liable to be unhealthy. A failure to achieve the full *repertoire* of functions which are the possession of the average mind is an obvious weakness, a want. But other minds, not to be termed imbecile, are also weak and unstable. These abnormal variations are brains in which one or a few functions are developed in excess, which means that the mind will not be well balanced. Such excess of function in one direction is apt to be associated with a lack in some other, and the mind fails to achieve that even activity under varying condition which is the sign of cortical stability. These odd minds are in my opinion much more deserving of study than cases of actual insanity. They repay study because, when short of insanity, they are more easily investigated; because they are of great importance in private life in that they are subject to endless petty disturbances, and are a great trouble to themselves and to their neighbours; and because they are predisposed to acute insanity, and ought to be recognised, understood, and guided.

Abnormal minds we shall therefore consider before proceeding to commonly recognised insanities. And the physician will find it useful to remember a somewhat arbitrary division of them into (1) Arrests of development, and (2) Disproportionate developments—abnormal mental variations or, briefly, odd minds. In most cases there are both elements. But in investigating the mental functions serially, the physician should note concerning each whether it is in excess or deficient.

Arrests of Development.—Arrest of mental development may occur in almost any degree and in regard to almost every cerebral function. We shall consider it as—

I. Partial.
II. General.
III. Progressive.

Partial arrests of development, as was said generally, occur in odd minds in association with excesses of development. One finds all forms of it, but we shall confine our attention to those which give trouble. Of arrested instinct, for example, that form is most important which is a lack of life-preserving activities. Obvious cases are those who simply do not care to live and do not fear to die. But a suicidal point of view is often safeguarded by a physical repulsion on the approach of bodily hurt. More important is a lack of instinct generally overlooked; in which the patient is indifferent to hunger and to fatigue. Many of our insane patients have been men and women who seemed never to know that sleep or rest was necessary, or that irregular fasting was dangerous. And many neurotic people show an analogous disregard of the safety of others. Normal children learn quickly what is unsafe; but many men and women never acquire a proper consideration for the safety of their neighbours, and do not react to a sense of danger to them. Then a great deal of vice and of misery is occasioned by persons who have not a well-developed sexual instinct. Many cases of unhappy marriage and many of sexual perversion are to be attributed to a lack of pleasure in normal intercourse. Like defects of organisation, though not with so obvious a bearing upon insanity, are maternal indifference, paternal disinterest in the home, irreverence or an absence of the instinct to worship, and the failure to eagerly defend one's country without respect to the righteousness of its cause, all of which wants are common in disorderly minds.

As we ascend the scale of mental functions, defects are of less importance from a biological point of view. We expect variations in the advancing points. But all the same some of them are of much practical importance. On the lower levels one of the most important defects is a lack of a capacity for enjoyment. Many good people take pride in their neglect of pleasures, but their failure to achieve a pleased interest in things sometime betokens a constitutional defect. No man who does not enjoy the really good things of life can be described as sane in the full sense of the word. Another primitive defect to which students of insanity should pay much importance is ineptness for muscular activities. Some of the most skilful in technique are neurotic persons; but very commonly those who are of defective brain cannot use their muscles aptly.

On a higher plane are to be noted such wants as we all are familiar with in the sphere of intelligence. Defects of memory are amongst the commonest; so also an arrested development of the understanding. Stupidity—an incapacity for thinking, and a disability in apprehending truth even when made plain—is so common as almost to be described as normal. Special importance attaches to such minds as combine some activity of thought with a lack of corrective judgment. A man who does not think is probably safer than a man who thinks well along certain lines, but who fails to see other sides of the question in hand. Rash judgments are very characteristic of defective minds. So also are truthlessness and a lack of common sense—a defective notion of probability and of feasibility. Odd minds are generally impracticable. Above all these, however, the student should carefully observe those who fail of social quality. The most obvious cases are those in which there is simple defect of sociability. More likely to end in disaster are those who, with a full development of sociability, are devoid of tact. Tact is one of the most interesting and subtle mental functions—a sensitiveness to the feelings of one's neighbours. And perhaps no one sign of the defective mind is so characteristic as tactlessness. Human evolution is towards a fuller and fuller understanding between man and man; and he who fails to realise that he is "rubbing his acquaintance the wrong way," the man who habitually says and does the wrong things, is singularly prone to mental trouble. Many persons commonly set down as bores or as egoists are of the insane diathesis. And lastly, we have to note those who with perhaps no want of manners are morally defective. Failure to develop self-control may almost be said to be a constant feature in odd minds.

General Arrest of Development.—Systematic arrest would, perhaps, better describe the conditions now to be considered. Partial arrest occurs apparently fortuitously in one or another mental function in persons who still pass in society for sane. In others, however, the mind as a whole has suffered arrest. Or at least there is a serial defect, most obvious in the last and highest functions, though with occasional developments of a disproportionate kind which seem excrescences in a weak mind. Two forms are to be noted: imbecility and degeneracy. Of these, imbecility is considered in a special article (*vide* "Mental Deficiency").

Degeneracy.—On this subject, which has become fashionable of late, a great deal has been said and written which will not stand severe criticism, and the student must take what will be said as a rather tentative account of degeneracy.

As I take it, the term describes a biological conception. A degenerate is one whose mind betokens a failure of his race. So far as he is concerned the race is coming to an end—not actually perhaps, but virtually. A degenerate may procreate children who will prolong his seed indefinitely, but if so, it will be by lucky marriage and in spite of his weakness. And such an individual is to be distinguished from the merely neurotic, many of whom are of a quite potent cerebral vitality. But if the practitioner sets out to diagnose degeneracy in its mild forms he will certainly fall into error. For the diagnosis between an arrest of mental function which is but a temporary interruption of a family's evolution and that which betokens the beginning of its end, is quite beyond us. And so in practice, at all events, we restrict our use of the term to obvious and gross types, such as are appreciable by an enlightened police. And that is fortunate, for gross degenerates are always immoral and probably always criminal. Dr. Urquhart has indicated the type. But a warning must be given because the subject has suffered a good deal from the amateur exposition of extremists who go much further than he has indicated. In particular, the stigmata, the defects of conformation in degeneracy, have achieved an importance which is altogether fictitious. From scalp to sole no spot or blemish escapes the category of the erudite observer. Degenerates have many of these blemishes; hence, it is argued, blemishes indicate degeneracy—a fallacy which a slight attention to biology, some elementary logic, or even a sense of humour, should have prevented. For we all have good friends who are very ugly; supernumerary digits do not betoken stupidity; early baldness or loss of teeth is not the monopoly of the vicious. The fact is, many of the stigmata of degeneracy are in more or less unessential organs which are undergoing the common fate of becoming rudimentary. As intelligence evolves it is quite likely that the race will depart further and further from a type of physical beauty which evolved centuries ago under the influence of direct sexual selection; and popular ideas of beauty are nearly as fickle as fashions of dress. Once upon a time, no doubt, the disappearance of fur from the human chest and shoulders was regarded as a grave event, and posterity will probably suffer hairless scalps and artificial teeth with equanimity. Surely these externals are of but slight importance, except for police purposes, compared with the internal processes which are essential to nutrition and recuperation. Sterility, susceptibility to infectious diseases, the tendency to other toxications described by Dr. Urquhart, cardiac and other visceral disease, anæmia, vascular degeneration, are much more significant of racial decay than are the anatomical features amenable to the foot-rule. The central fact is, of course, that in degenerates these defects of physique, both external and internal, occur in abnormal degree.

Of mental facts, immorality is the primary and essential feature of degeneracy. Morals are, at least theoretically, the budding point of evolution, and no one may be regarded as degenerate who feels and knows and does the right. And to degenerates lack of morality usually means criminality, for most degenerates are too odd to be law-abiding, and too stupid to escape. In the ranks of society higher than the lowest, however, the temptation to commit police offences is not so great, or, as in the case of sexual perverts, detection may be very difficult. But failures other than in morals are always to be noted in the degenerate. He may be clever, but not of sound judgment; very knowing, but not wise; fond, but not faithful; sentimental, but not just; convivial, but not happy; active, but without endurance. And the practitioner must be prepared to recognise these potential criminals, whether noble or lowly born. His common knowledge of the human features is all the anthropology that he requires for practical purposes; he will pay great attention to facial expression; he will note carefully any defect of nutritional processes; and, above all, he will try to discover, unostentatiously, the feelings and ideas and impulses of degeneracy. And, having assuredly diagnosed these creatures, he will treat them with a view to the safety of society, and with just as much leniency as is feasible. For these unfortunates no liberty, no opportunity of evil, is safe. They must be controlled and disciplined, segregated or banished, or otherwise treated as rigorously as circumstances justify.

Progressive Arrest of Development (Primary Dementia).—The cases now referred to, combine arrest of development with active degeneration. They are cases in which the mind develops up to a certain point and seems to the patient's friends to be going on well. Then it stops, and forthwith deteriorates more or less rapidly. Such cases of apparently idiopathic dementia occur at all stages of life, and they comprise a most interesting group not easily understood. To explain them we must revert to the idea of a premature senility of nerve-cells which inaugurates something in the nature of a descending degeneration. For example, take the case of a boy at school, a good son and a promising scholar, who, on entering the fifth form, failed to make progress, and, at the end of the term, returned home with his first unsatisfactory report. A year later that boy was obviously demented, had apparently forgotten all he had learned, and was fit only for the work of a common labourer. Some such cases go on to motor paresis. I have under my care now a man who broke down towards the end of his college days after an unusually promising record. There was no sudden change, but only a slowly progressing stupidity, and instead of taking the high place in his profession which was expected of him, he became entirely childish. Moreover, he suffers from a partial hemiplegia, with diffuse motor impairments in all his limbs. His attitude and gait, and especially his speech, are those of an idiot. Visitors who are specialists take him for an imbecile, and strangers cannot understand what he says. Yet there was no sudden lesion, and in my six years' acquaintance with him, his atrophies, as we call them, have not materially increased. Most careful inquiry failed to elicit any suggestion of an active cause of insanity.

Some of these cases are occasioned by the reproductive crises. Precocious senility may occur after child-bearing, or with the too heavy cares of a large household. Similarly, men who become rather suddenly engrossed with large and increasing affairs may not only not rise to the occasion, but may become demented under their burden. And so we can trace the disease right on into recognised senility. Healthy brains are still plastic in old age. Some men and women never know dotage, but others begin to be senile at the climacteric; and, from that time onwards, instances are very common of progressive mental atrophy, with or without sensory and motor impairments. Though we find it expedient to consider "Consecutive Dementia" separately, those cases of acute idiopathic insanity, followed by dementia instead of recovery, are probably cases in point, and their acute attack merely an incident in the atrophic process. Some of them, after an indefinite period of dementia, undergo a more or less rapid motor paralysis from diffuse degenerative changes. Americans, under the name "Hebephrenia," describe a disease which occurs at or soon after puberty, which may include an acute attack of insanity, which leads to craziness and dementia in about a year. It occurs in lawless patients, and masturbation is a frequent factor. It would therefore appear to be an abrupt progressive dementia in the obviously degenerate.

ABNORMAL MENTAL VARIATIONS.—Alienism suffers greatly from a defective nomenclature, and especially from the misappropriations of generic terms to a too narrow application. Thus, degeneracy, which ought to describe mental arrest in general, is used almost exclusively in reference to the extreme, criminal cases. Similarly, the term paranoia would have been most suitable for the designation of constitutionally disordered minds not actually insane, but is used almost exclusively to describe a delusional variety. We must therefore fall back upon a roundabout phrase—abnormal mental variations—to describe the symptoms of those odd minds which are of the insane diathesis. To gather our threads together, we may recall summarily the most important wants of such minds. These are a lack of life-preserving instincts; defective sexual desire of a normal form; domestic indifference; lack of capacity for enjoyment; muscular ineptness; gullibility; unpracticableness; and tactlessness. These are the negative signs of the insane diathesis, not all of them to be looked for in one case. And associated with them we must note many positive symptoms—disproportionate developments of function which add to the mental asymmetry. Their name is legion, and we must confine ourselves to a few of the most important.

Of the primitive functions, the sexual claims first attention. History teems with instances of redundant sexual capacity of a normal form. And more significant are the various perversions of it, so hard to explain except as due to disarrangements in embryo. Of the other instincts, all manner of excesses might be described, but other kinds of disorder are more significant. Very important, for example, are disorders of sensation. A visceral hypersensibility is often to be noted, and still oftener, an undue attention to stimuli of the special sense organs. Similarly abnormal minds may manifest undue muscular aptness and develop extraordinary movements. The best instances are those musical prodigies whose technique is so far in advance of their feeling for music. But it is on a higher plane that we find the best symptoms in point—in great feats of memory, in the tenacity of fixed ideas, in extravagant beliefs, in too subtle thinking, in unbridled imagination. Of the last, day-dreaming is very important, as visionary activities of that sort are often the prologues of delusion or of insane impulses. On the emotional side of mind we must take special note of fearfulness, that relic of the fugitive consciousness which expresses itself in an unfounded apprehension of impending evil, a tendency to expect an evil issue out of every experience, a habit of "supping sorrow with a long spoon." No special kind of emotion, however, is characteristic, but rather an undue intensity of whatever feeling the mind entertains. In volition, impulsiveness is the most common excess. In the ill-balanced mind promptings of an imperative kind, and which are not justifiable by the facts of experience, call for a control which is often lacking. And lastly, extravagant conduct is characteristic—a manner and behaviour which is on the way to the ridiculous, or which sails very close to the wind and verges on the disreputable.

As I have said, these signs of abnormal variation may be multiplied indefinitely, and from what has necessarily been little more than a list of the more important, the physician may gather little that will be of use in practice. It remains, therefore, to refer to a few of the more frequent types of persons who manifest the insane diathesis; but always with the premise that even those who seem to conform most closely to such types are not to be set down as abnormal unless there is an abundance of contributory symptoms. But many of the symptoms which have been mentioned will be found in some of the following:—

There is a type of men and women who are the impersonation of timidity—shrinking, cringing, diffident, bashful—whose knees seem to give way on the slightest provocation, who are always afraid, but who may be all the same capable of great and even of heroic things. There is the sentimentalist who wallows in feeling, who cannot take a cool and reasonable view of anything,—pious perverts who translate everything into the terms of a silly religion which finds almost no expression in a dutiful life; devotees of æsthetics, who sacrifice everything to their sense of beauty; and other sensuous varieties. There is the amorous type of persons who take the opportunity, in season and out of season, to engage the affections of their promiscuous victims; feminine men who dote upon male beauty, masculine women who fight shy of men, the sanest of whom find expression in asserting the rights of their sex; and others who make a fetish of prudishness and "dwell with inverted gusto" on the evil of a feeling which, in its normal form, nature has denied to them. Of another form are the grievance-mongers, persons of an inveterate suspiciousness, always smelling a rat, ready to take offence, hopeless as wives or husbands or colleagues in office. There are the half-insane snobs, devotees to "good families," toadies, and sycophants; and the "affiliative" variety who, on the slenderest grounds, trace their relationship with all and sundry who can be of use to them.

Then there are the recognised paranoiacs, victims of disordered thinking, many of them more or less degenerate. The generic feature of them all depends upon a temperament which indisposes them to get on smoothly with their neighbours. The classical type is the querulous paranoiac, a variety of grievance-monger who airs his disputes in public, and supports his case with obviously deluded arguments, who can never take a decision against him, carries his case perhaps from one court to another, and who often has recourse in the end to a violent attack upon some personage of note in order to attract the attention of an indifferent public. In private life the tactless bore is almost as grievous a person, full of superficial ideas, inordinately pleased with his sickly wit, quite oblivious of contempt, not easily snubbed. Then there is the gull, the butt of all his acquaintances, good-natured often and often secretly vain, whom any one can impose upon, and who never learns from experience. And Paul Pry is another type, the curious paranoiac, whose nose is always in his neighbours' affairs, or who, in a less harmful sphere, buries himself in useless investigations. Then there are the unpersuadables, whose ideas are hopelessly fixed and who cannot see the force of argument, or, if they do, "are of the same opinion still"; and all the cranks and fanatics, "idea-fiends," as America knows them, who find in teetotalism, anarchy, socialism, or a land-tax, the solution of all the world's difficulties. Lastly, there are the more or less thoughtless persons whose conduct is disorderly. Of these, we may note the dare-devil, who does anything he cares without apparent regard to safety; casual paranoiacs who cannot appreciate the importance which men attach to their time and to their property, careless, haphazard, unpunctual people who break their engagements and do nothing thoroughly or exactly; gushing men and women, voluble in expression, silly and empty-headed, except for a store of useless information; the would-be reformers who never see anything they cannot improve upon; and the feckless, who can never arrange their affairs, who spend their days in vain efforts to redeem lost time, always busy and never doing much.

These people, ill-nourished, or prone to excess, or careless of health, often come to an unhappy end, perish in some hopeless cause, pamper themselves with the connivance of foolish relatives into chronic hypochondria, or, driven from pillar to post, from post to pillar, end in an asylum. A little care and systematic inquiry easily furnish the facts by which to recognise the more precarious of these variations. We must not, however, conclude hastily that a man is of insane predisposition merely because of some conspicuous eccentricity or bad habit. From such judgments has arisen the preposterous fallacy that geniuses are usually degenerate. As has been noted, some abnormal people are prodigiously accomplished in certain directions. But the common sense of mankind revolts against the judgment which classes with them the really great minds of history. A Shakespeare, a Goethe, a Napoleon, does not manifest arrest of development, and a woman must needs have suffered much from modern teaching to be afraid to have such an one as the father of her children. These great minds apprehend much that is beyond the common perception, but their genius was based upon a strong fabric of primitive human instinct. Other kinds of genius, still not necessarily degenerate, are instances of extraordinary developments of some special functions of the intelligence, such as the inventive, which do not imply an all-round sufficiency. Such minds *may* manifest an arrest of one or more of the important mental functions, and in that case insanity is more likely to overtake them. Physiologically speaking, genius is simply an unusual vitality of nervous mechanisms whose function is such as fits with the environment of the time. The same kind of account must be given of "strong men" (muscular geniuses) as of a great poet. But the sane genius is one whose extraordinary gift is the accomplishment of a mind which is fit for all the essential functions, and apprehends all the essential traditions, of his race.

Further allusion to this subject will be found in Dr. Macpherson's article. For these odd minds are apt to end in delusion, or to suffer that combination of degenerate feeling and degenerate thinking which constitutes Paranoia (*q.v.*).

These abnormal variations which we have considered call for prophylactic treatment such as Dr. Urquhart has suggested, and a great future lies before mental science in this application of its knowledge. But it will not be until the public realise that the brain is an organ capable of scientific management, and not until parents and practitioners habitually observe and study mental facts, that we shall enter into this, the only hopeful field, of mental medicine.

IV. The Incidence of Episodic Insanity, with Special Reference to Bodily Signs

As an episode in the life of man or woman, whether of the insane diathesis or no, insanity is nearly always ushered in by characteristic signs, and there is no aspect of the subject of greater importance to the physician. Again and again he will be face to face with some one who is in very bad spirits, or unduly exalted, or expressing some strange beliefs. Is this person then merely depressed or joyous from some passing cause, and are these strange beliefs merely mistaken but intelligible opinions? Or is there here the incidence of an insanity—the invasion of the organs of personality by a disease which may or may not be checked, but which in any case is sure to be grave? And these questions should be promptly answered, for a large majority of the insane are curable, and early diagnosis is of the first importance.

Speaking generally, nearly every case of insanity is attended by a more or less profound disturbance of all the important bodily functions; and the more grave the insanity is going to be, the greater that disturbance is. Before acute insanity the following changes are to be noted:—

Nervous System.—Disturbance of sleep is very common, either as sleeplessness or as dreams, much more rarely as extreme somnolence. In melancholic insanities anergia is usual—a disinclination for exertion and an oppressive lassitude. In mania and in delusional insanities the patient has often manifested restless energy. The muscular system is generally irritable, and tremors, slight spasms, and exaggerated muscle reflexes are almost invariable. A great number of cases complain of hyperæsthesia.

Digestive System.—Digestion is almost invariably disturbed, and loss of appetite is nearly constant in melancholia. Dyspepsia is associated with constipation, which the patient is apt to ignore, and the tongue indicates it.

Circulation.—In every case in which melancholia or mania is imminent a change in the character of the circulation occurs, but there is not always a characteristic change. In both diseases the pulse begins to be variable and excitable and fickle in rhythm. There may be hypertension or hypotension in either disease, just as in sane joy or grief. The important point is, as Dumas proved (*Revue Philosophique*, June, July, August, 1896), that the circulatory change always precedes the mental. He describes a case of alternating insanity in which he was able to predict the attacks from the pulse. There may or may not be stasis and congestion. Œdema and asphyxia of the extremities is common in lethargic melancholias and in stupor.

Changes in the constitution of the blood have often been observed, but the practitioner is not likely to know what any given case normally manifests in this connection.

Urine.—We may say of the secretion of urine, as of the circulation, that when insanity is impending there is usually a marked change, but that the change is not of a distinctive kind. Perhaps the commonest fact about patients before becoming insane is that, instead of an equable secretion, they suffer from a diminished quantity, with high colour and high specific gravity, with periods of polyurea in which the urine is pale and of low specific gravity.

General Constitutional Changes.—These are most important. In a large majority of cases the patient has been losing weight. Very rarely is the incidence of insanity coincident with increasing weight. There are two great types of constitutional change to be noted. In one set of cases the patient is observed by the relatives to be growing thinner, paler, more wasted and haggard in appearance. In another there is an appearance with which I always associate the word "surfeited." To the eyes of relatives the patient appears more gross—not necessarily gaining weight. The complexion becomes muddy, the expression heavy, the eye dull, and the patient himself may complain of feeling "clogged up." Perspiration is usually very scanty, but the skin may be greasy. This appearance of surfeit is unmistakable, indicates a defect of excretion, and probably denotes toxæmia.

Reproductive System.—Menstruation is almost invariably disturbed before insanity and the sexual function modified.

The *mental signs* of impending insanity need not be enlarged upon. However, it may be noted that, except in the rare cases of almost instantaneous onset, the patient's habits invariably change before an attack; the domestic, personal, and business traditions are departed from. That is much more significant than any actual feeling or idea expressed by the patient. If the state of mind which excites the physician's suspicion has not been preceded by altered habits, there is almost certainly no acute attack impending unless there are some extraordinary circumstances to cause it, a disaster or other great shock. We must therefore inquire carefully into the circumstantial facts in every doubtful case. In an interview with the patient greater importance attaches to facial expression and to manner than to what he says and does. Before insanity has become actual, patients almost invariably change in appearance, and express preoccupation, suspicion, or dread, even if they do not admit it. At the same time the face assumes an unwonted brightness or dulness.

In every instance it is a change from what is normal in the patient's case that is significant. Nearly every function which I have specified—both mental and bodily—will be found to alter when acute insanity is coming on, and the physician who systematically investigates his case will very rarely be taken by surprise. When the attack has begun, and during its course, these bodily symptoms become aggravated. Derangements of a constitutional kind and disorders of one or other of the systems are almost invariable in episodic insanity. But the symptoms are so variable that we shall not enlarge upon them in the following account.

V. Types of Episodic Insanity

Having glanced at the permanent abnormalities, we now consider acute insanity as it appears as an episode in the life-history of a mind. Sometimes these actual insanities occur in persons such as have been described as of the insane diathesis, and in that case the disease may be traced as a gradual development of habitual modes of thought and of feeling. In other words, a constitutionally abnormal personality may become more abnormal and cross the line beyond which the patient may not be regarded as responsible or as fit to take charge of his life and affairs. Or some accessory cause or some crisis in development may lead to a gross cerebral disturbance, which becomes evident as a definite and well-marked departure from the usual mode of consciousness. And that is what always happens in the case of persons who, though not of a recognised type of abnormal mental variation, suffer an episodic insanity. From any of the causes, except inheritance, such as Dr. Urquhart has enumerated, a fairly orderly mind may suffer a cerebral attack, and such an insanity is an invasion of the personality, slow or sudden, under which the normal character may disappear and give place to feelings and beliefs and conduct which are quite alien to it. That is, subconsciousness may usurp personality. And it must greatly enlarge our conception of the subconscious to study types of insanity. For the very fact that there are such types implies that the human brain, on a plane a little lower than that which becomes realised in sane life, is still a more or less constant and systematised organ. To my mind that is a very important conception, and one which is perhaps not realised by all physicians who concern themselves with mental functions. In a lesion of the motor area with partial paralysis the abeyance of movement is not followed by indefinite incoördinations, but by secondary symptoms which are systematic. If the lesion is unilateral, muscles of the sound side produce an excessive effect, as in pushing the tongue or drawing the mouth aside. Similarly, if the lesion is high up, the descending order follows quite definite and systematic paths. The spastic gait or that of tabes is characteristic because it is more or less constant, because the excessive contractions in the surviving muscles depend on the systematic arrangement of the surviving mechanisms. An exactly analogous system or organisation pervades the lower mental areas, so that, when personality fails, the brain does not manifest sheer disorder, but usually reveals a typical activity of surviving mechanisms. What type it will be depends upon constitution and upon the seat of the lesion. The best example in point is that of alternating personality (double consciousness), a most interesting and not uncommon form of insanity. In that disease, when the first personality subsides, the cerebral activities are determined by mechanisms of a second personality which were, so to speak, waiting in readiness for their opportunity. But though not so obviously, every insanity reveals an analogous orderliness. Even acute mania is not really a disordered but a quite systematic over-activity of sensori-motor mechanisms. Were these facts not so there would be, as I have said, no types of insanity, and the insane would be perhaps weeping because they were millionaires, or committing suicide in order to get the crops in, or making homicidal attacks on all and sundry because tables should be made of glass. Such real incoherence is extremely rare. Moreover, the types of insanity which we do recognise are for the most part secondary symptoms. They are analogous to a description of motor paralysis in terms of the contractures and hyperæsthesias of the surviving mechanisms, as Hughlings Jackson long ago pointed out. Acute insanity is a greater or less degree of failure of normal personality with all that that entails. The primary lesion is in the mechanisms of sane character and sane conduct which have gone by the board, leaving an activity in lower mechanisms by which we designate the disease.

It has been suggested by many authorities—Bevan Lewis, for example—that clinical varieties of insanity are not to be regarded as essential, that they are "purely artificial or arbitrary divisions," that the pathological process, vascular or nervous, is the important thing. The practitioner, however, wants first to know what the clinical developments in any given case are likely to be, and he is right in insisting upon that knowledge. For in all nervous affections the site of the lesion is as important as the lesion. It is important for our knowledge of disease to distinguish between thrombosis and glioma, between apoplexy, embolism, and sclerosis. But in each case the consequent impairments are part of the illness, and what they will be depends upon the site and size as well as upon the nature of the lesion. And these things are still more true of the lesions of insanity. We may safely defy any one to predict the differences in the course of a mental disease which is due to slow vascular obliteration, and one which is of the nature of a slow neural atrophy. The differences that we recognise are chiefly differences in the positive secondary symptoms. Abeyance of normal personality is the prime fact in all episodic insanities. Thereafter the course of the disease depends upon what groups of lower functions become too active, and that is an affair of anatomy—a topical factor. That experiment and disease have localised and demonstrated the Rolandic centres does not make them more real. Hughlings Jackson and others knew of their existence long before. And similarly, recognising as we must a domestic mode of consciousness, for example, a fugitive, an amorous, a constructive, we are bound to infer a systematic mechanism for them; and our knowledge of it will not be greatly enhanced even if, as seems unlikely, some one should demonstrate their position and form. There are exceptional cases, but the vast majority of insanities are only intelligible as systematic secondary affections. Were consciousness and its mechanism not systematic there would be, as I have said, no clinical types of insanity.

Melancholia.—Melancholia is characterised by an abeyance of interest in things in general. That is the primary fact, the *negative* symptom. Following that loss of habitual conduct and of interest, the patient becomes self-absorbed, dwells upon his misery, seeks to find some explanation for it, or perhaps tries to put an end to it by insane violence. In any case he cannot forget his pain, and that self-regarding mode of consciousness is the secondary, the *positive*, fact in his case, and it is constantly present in melancholia. Moreover, this mode of consciousness is not fortuitous. It is determined by subconscious system. Self-regard to an insane degree is provided for by inherited cerebral mechanisms, and would predominate in all human minds but that higher interests and activities overrule and prevent it. Melancholia is the simplest, the most likely result of insanity. Its primary features are, as I have said, a loss of physical energy, an anergia, so that the patient is not fit for usual activities, and a loss of interest in life, so that the mind does not attend to affairs. A very slight investigation will reveal these facts. A melancholic is habitually tired, and if compelled to undergo great exertion will certainly become worse, and probably proceed to mania or to dementia. But the exhaustion is most notable in the highest functions, such as are normally concerned with the conduct of life. Hence, as Dr. Mercier points out (p. 384), the direct benefit of a complete relinquishing of all responsibilities and duties. In many cases the nervous depression is due to bodily disorder, but at the same time the failure of the functions of personality induces or occasions physical deterioration, and we can never exactly say how much of the depression of bodily functions is a cause and how much an effect. The fact remains that a little inquiry will reveal disorder in nearly every system—dyspepsia, constipation, and anorexia, a variable heart, a soft pulse, cold, and perhaps swollen extremities, diminished secretion of urine, dry skin and hair, a loss of weight, sleeplessness, muscular irritability and flabbiness, diminished surface sensibility, exaggeration of deep reflexes, and often neuralgia, stiffness of muscles, and difficulty and slowness in executing fine movements.

The loss of outward relation is the central and primary impairment of mental function—the abeyance of object consciousness, as Bevan Lewis designated it. The melancholic is interested in nothing and in nobody in his neighbourhood. He neglects duties and pleasures alike, and forsakes his habitual occupations. When the disease is advanced even striking events pass almost unnoticed, and emergencies induce no response. That of course betokens a serious failure of personality. Thereupon lower forms of nervous activity become apparent, chief of which is the self-regarding mode of attention — the rise of subject consciousness. That is one definite phase of subconsciousness. The mind, on relinquishing usual affairs, might become occupied with the unusual, as on a holiday, or in delusion, or in mania. In melancholia it becomes occupied with itself;

and a persistence of that form of consciousness is inevitably associated with unhappiness. Pleasure is the content (the subjective fact) of activity for which there is ample cerebral energy. The conditions of pain are inactivity or activity in excess of vitality, both of which are present in melancholia. The exhaustion from which the melancholic suffers occasions weariness and distress, and his inward regard perpetuates it. All the rest of melancholia is the attempt of the brain to give expression to its altered activities. It is probably a misuse of terms to speak of the melancholic personality, for when the old personality has gone—we cannot say whither—what remains is not worthy of the name, but we may call it the melancholic or inward character. And that expresses itself in various ways which determine what are somewhat arbitrarily discriminated as varieties.

SIMPLE MELANCHOLIA.—In *simple melancholia*, for example, outward movement is of the slightest. The patient is simply and monotonously depressed and quiet, does not moan much, or otherwise express grief. The intelligence may be fairly active and the patient able to appear in society without attracting attention, though always with a burdened mind. When alone, however, he probably resigns himself to his pain, and already has glimmerings of delusions such as might seem to him to account for his distressed state. The characteristic fact of this mode of consciousness is this constant undercurrent of pain—a disorder exactly analogous to exhaustion of any peripheral part, though, for reasons referred to in the account of the mind's relation to the brain, the patient does not refer his pain to his cerebral mechanisms, does not locate it. The condition is what is known as a systemic sensibility of an unpleasant tone. In health the sum of cerebral activities has a balance of energy on the right side, in consequence of which the systemic sense is pleasurable, and creates a joy in life. The melancholic, on the other hand, does not find it good to be alive, and from a very early stage would not be sorry to die, and may even feel promptings to end his misery by suicide. And that undercurrent of ill feeling can nearly always be diagnosed, even if the patient will not plead guilty to it. If you watch him unobserved, you will see him assume that indescribable look of self-absorption, as, perhaps, in the middle of some occupation he turns his mind inwards with a sigh, and from vacancy his expression will pass to one of actual pain. Moreover, melancholia has what is called a gymnastic—a definite muscular state, just as exaltation has. Over and above the slight degree of atonicity which prevails, and which occasions attitudes and movements that betoken slight muscular feebleness, the voluntary muscles assume positions of automatic contraction. The characteristic pose of exaltation is in general one of extension. But you will rarely observe extension in a melancholic unless he is making an effort. When he lies down his legs automatically flex, and his arms and hands. Similarly his brow puckers downwards, his mouth purses, and his eyes are rarely wide open. I am speaking, of course, of what happens when the patient is not aware of his movements. In other words, the muscles are beginning to assume the poses appropriate to grief, and which in their extreme form are known to us all as revealed by the actor's art, or as commonly expressed in sane woe. Other varieties of melancholia similarly suggest that the disordered activity follows a systematic course.

ACUTE MELANCHOLIA.—The progress of disease from simple to acute melancholia is obviously systematic. It is in fact merely a deeper degree of melancholia. The outward regard, occasionally possible in the simple variety, is now wholly impossible. Self-regard correspondingly increases, and so also does its accompanying pain. Similarly the gymnastic of melancholia is intensified, and the patient's muscles automatically assume poses and attitudes which are stereotyped as well-marked expressions of distress. (Note that, except on the stage, these shows of grief are rarely observed in the sane.)

In acute melancholia volition is profoundly affected, so that the patient is no longer able to conceal his grief, and with every new loss of power self-regard is accentuated and its consequent pain. Or to express it more correctly, outward regard is still further lost, and nothing in the environment has its proper influence on the consciousness of the patient. In many cases this is so pronounced that even if you speak to him, or try to move him about, he behaves as one in a painful dream, not looking up, not speaking, apparently never turning his attention from his distress. And it is important to observe that this excess of self-regard is without adequate reference to normal bodily facts. For while in the visceral variety of melancholia there is over-attention to internal stimuli, the consciousness, in simple and in acute melancholia, does not sufficiently embrace ordinary physical facts. Attention to what we may in a very wide sense describe as the "calls of nature" are inhibited. The melancholic seems not to be aware of the discomfort of an empty stomach, of a full bladder, of drenched garments, of an unwashed and unkempt person, or even of minor surgical injuries. And much of our treatment must be a provision against the dangers of such a state. The motor signs are similarly accentuated, and the classical expressions of pain sit, from morning to night, on the patient's face, hands, shoulders, trunk, and legs. And now also the morbid activities find their way in systematic directions much more obviously. There is much more mental movement of a disordered kind. For example, instead of the intervals of sane thinking of which the simple melancholic is capable, and which alternate with perhaps simple attention to pain or vague surmises as to its cause, the patient becomes possessed with an emotion which he explains by means of delusion. And that emotion is nearly always to be described as fear. Acute melancholia reveals the survival of the fugitive consciousness. The patient is pursued by fear, very often in the form of remorse, terror at the thought of the possible consequences of the wickedness or folly of which he imagines himself to have been guilty; or his panic takes a prospective direction, and he is constantly expecting a dreadful misfortune; or he believes—which is most likely in those cases in which there is some ground for it, and which is occasioned by visceral sensations—that he suffers from incurable disease. Now also the disorder may be observed to spread towards the special senses, and attention, finding no foothold in sane and outward activities, becomes possessed by suggestions which emanate from the visual areas, or more often from the auditory. In the beginning this is evident merely as hyperæsthesia. The patient hears far too much. Or, perhaps, we would express it better as over-attention and say that the patient listens far too much. And that happens which we might expect if the disease is not stayed. Watching and listening for sights and sounds instead of attending to affairs, the patient will become the subject of hallucinations, at first vague and irrelevant, but later appropriate to his distress. For here, again, we have to note the systematisation of the disorder. We cannot suppose that in this state of disordered perceptual activity only appropriate hallucinations occur. But the distressed attention selects what is relevant, and, as always, attention to the distressful increases the facility of their occurrence; and so painful hallucinations survive over others. All these are the positive survivals of activity in acute melancholia. In this condition purposeful acts are almost in abeyance. But there is still the risk of suicide of an impulsive kind, though the patient is now unable to plan and to scheme for it.

ACUTE DELIRIOUS MELANCHOLIA.—Acute delirious insanity generally takes the form of mania, but not always. As has been hinted, the prime difference between mania and melancholia is the survival of outward activities in the former. In acute delirious melancholia these movements are not found, but other symptoms of it are present. In some cases of acute melancholia the signs of bodily disorder become very prominent. In particular, some patients take on what I have described as the bodily signs of surfeit so suggestive of toxæmia. And that may go on to an extreme state in which, with diminished or altered secretions, furred tongue, sordes about the mouth, dry skin, rapid pulse, rise of temperature, and prostration, there is the apparent unconsciousness of surroundings and of the self which is characteristic of delirium. In this stage then the primary, *negative*, fact—the lostness of the sane personality—is more than ever obvious. So also the secondary, the *positive*, symptoms are on an ever lower level of cerebration. The patient has ceased to be simply self-regarding and distressful. His conduct, and presumably his consciousness, are of the delirious mode, as in typhoid or in pneumonia. Even the gymnastic of pain has gone, and the patient's muscles move automatically in delirious movements—chiefly muttering and picking. In all probability the consciousness now is barely capable of description. It would appear to be occupied by flitting sensations and by quite vague and dream-like ideas. The over-activities with which it is engaged are apparently in the Rolandic area or below it—unrelated disturbances of motor and sensory mechanisms of a primitive form.

DELUSIONAL MELANCHOLIA.—Simple melancholia, if not cured, may go on to an acute form; or the acute or the delirious variety may develop very suddenly without passing through that stage in which the depression is a background to a life still capable of outward activities. On the way to becoming chronic, nearly all melancholias are delusional. Such a development is probably less painful; for, obviously, when the patient is constructing an argument to explain his state, he is not entirely self-regarding. Delusions are, of course, *positive symptoms*—the wandering fancies of a "lost mind," which betoken activity of a secondary kind in the now-surviving mechanisms of intelligence. And here, again, we must note that the melancholic mind abides by fairly constant paths. Nearly all melancholic delusions refer to one or other of the primitive instincts or traditions of the race; and, having taken a line, each patient generally abides by it for a considerable period. It is always a good sign when the subject of the delusions alters. When the consciousness departs from one set of delusions it is quite likely to be on the way towards recovery. The most usual types are as follows:—

Consciousness of Immorality.—Many patients are convinced that their distress is the consequence of their evil deeds. And not infrequently there is some truth in the idea. A large number of patients who believe that they are eternally damned have been the victims of a vicious habit, and a bad conscience is a potent cause of insanity. A considerable number of very righteous persons, however, believe when insane that they have been horribly wicked. As a rule the belief is vague. But sometimes the patient conceives a concrete crime—murder, or infidelity, or theft—and harbours the delusional belief that he has been guilty of it.

Consciousness of Persecution.—Delusions appropriate to a fugitive consciousness characterise a form of slow insanity which we shall consider later. But many melancholics also, bowed down with pain, conceive the idea that they are the victims of a conspiracy, or wanted by the police, or pursued by an angry God, or played upon by electricity, or undergoing slow poisoning. These delusions in such cases are not abiding.

Consciousness of Social Disaster.—Many patients on retiring from society under their grief become possessed with the idea that they are disgraced in the eyes of their neighbours, that people look askance at them, that they are defaulters and ruined. Melancholics who seldom manifest an interest in outside affairs will sometimes devote themselves to the newspaper to an extent difficult to understand until we learn that they are looking to find themselves in one of its black lists. Similarly, when a patient is removed from home a very common delusion is that a domestic disaster has occurred, that the home has been burned down, that the spouse is dead or has fled, or that the children have been murdered.

Consciousness of Bodily Disorder.—These classes of delusion are not discrete, but obviously overlap. No description of mental facts is or can be exclusive. And many delusions, otherwise classified, are occasioned by a consciousness of ill health, whether interpreted as such or described in other terms. That mode of attention is sometimes general—does not refer to any particular function or part of the body, and then the patient probably believes simply that he is dying, or labouring under some incurable unspecified disease, or emitting an offensive odour. Or it may be particular—refers to some one system or symptom—and then perhaps the patient believes that insects are crawling over him, that his stomach is of glass, that he has cancer of the throat, that he has lung, or heart, or abdominal disease. The systemic ill feeling such as occurs in neurasthenia or in toxæmia probably accounts for this hypochondria of a general kind. And we call it hypochondria, not because there is not actual disease, but because the patient suffers the invariable hypochondriacal symptom of being wholly unable to ignore his trouble, and, not content with feeling and thinking about it, discourses freely upon the topic. Particular delusions are more often based upon local disorder. Hyperæsthesias and anæsthesias probably account for many such beliefs. Visceral delusions often prove to be approximately true, and physicians do well to make frequent examinations of patients who insist that a specified function is disordered. In particular, as Dr. Macpherson, in this country, and others have recently insisted, we must have great regard to indigestion or paradigestion in the insane. The belief of a melancholic that he has an animal, an embryo, a clock, or a battery "in his inside," is often a misinterpretation of a very real fact. A large proportion of melancholics are dyspeptics and have dilated stomachs. Many hours after a meal, if vomiting occur, or if the stomach be emptied by the tube, undigested food will be ejected. Hence the modification of the old idea that melancholics should be indiscriminately stuffed with food, and the advantages in certain cases of washing out the stomach, and, as Dr. Mercier suggests, of a strictly regulated diet.

Suicidal Melancholia.—If melancholia occurs, as it often does, in a person who is lacking in the life-preserving instincts, he will certainly become suicidal if his distress is considerable. We can appreciate what it means to him if we consider (what is a fact and not merely an expression) that a victim of a bad sea-sickness really does not care whether the boat goes to the bottom and he with it or not. The distress of melancholia, and probably of stupor, is what we would describe as intolerable. Most cases would be glad to die, but most cannot bring themselves to perform the suicidal act. The prolongation of life is very largely a muscular affair. The life-instinct is to a large extent an inherited series of self-defensive movements—to draw a limb away from hurt, to run from danger, to shrink from inflicting a wound on oneself—all of them as automatic as the blinking of the eyelids in a dust-storm. In suicidal melancholia one of two elements, or both, may be present. These instincts for danger, the common-sense appreciation of what will hurt, may be not fully developed, or the patient may have great pluck or determination, hence the discrimination which patients show as to the mode of suicide, and the discrimination which the physician must exercise as to the fatal probability in a given case. The only safe assumption is that each and every melancholic will be glad to die, and therefore it is never safe to leave them unguarded in circumstances which offer, and perhaps obtrude, an opportunity of suicide. But the variety of melancholia which we are now considering is a much more positive state. It is not merely carelessness of life, but a fixed and persistent determination to put an end to it. Such cases are not infrequent. They are not of the acute form of melancholia, which has almost no outward relation, but generally speaking have considerable mental activity, probably converse a good deal with their neighbours, and are able at times to quite conceal or forget their distress. Their state of mind is like that of simple melancholia with a suicidal intention added. They are like persons who know they must die soon, sometimes are cast down by the thought of it, sometimes mercifully forgetful, sometimes deliberately deceitful about it. Every such case has an insane conviction on the subject—that his or her death is necessary or expedient. Sometimes that conviction grows in a mind which does not reveal much other insanity, but quite as often is an insane conclusion from insane premises. Such patients believe, for example, that they are a source of danger to their neighbours, that the world will not go right till they are dead, or that they must die to expiate their crimes, and not infrequently the grounds for bringing about their death are furnished by hallucinations. From that of the religious fanatic who hears God calling upon him to come away from the earth, to that of the degenerate criminal who is beset from morning to night by fierce voices which cry out upon him that he must be slain, there are endless forms of hallucinations such as may promote the suicidal determination.

In suicidal melancholia, then, the systematic nature of the disease is obvious. It probably occurs in people who have not a normal fear of injury and of death. This lack of life-preserving instinct is an inherited quality of a negative kind, and when useful activities of an outward kind become impossible and pain supervenes the consciousness assumes the suicidal mode. It is fortunately rare, for all pronounced melancholics would be suicidal were it not that the vitality of the life-preserving instincts is enough to withstand and inhibit suicidal suggestion. But they all suffer some degree of abeyance of these instincts in their disregard of the calls of nature to which I have referred. No acute melancholic takes pains to live, though only a few take pains to die.

HOMICIDAL MELANCHOLIA.—Just as the melancholic's distress may find a systematic expression in a determination to commit suicide, so also may it find an outlet in homicide. Here again the apparently disorderly activity is one which has a definite place in the patient's consciousness. Just as he might believe his own death important, and bring it about, so also he may come to think that some one else should die, and attempt to achieve that. Sometimes these suicidal and homicidal intentions are associated. Hence those tragedies in which a father kills his family and then perhaps himself. In some cases the homicidal melancholic entertains a delusional belief about people in general, such as that the more people he can kill the better God will be pleased. Sometimes he believes himself called upon to extirpate a class of persons—the profligate perhaps, or the oppressive—and in his weak thinking he may attack persons who are not of the class referred to. More often the grounds of homicide are personal, and the patient attacks persons whom he believes to be in conspiracy against him or otherwise the medium of his misery. And then also the signs by which he recognises those whom he considers his enemies are generally fanciful and slender. Lastly, he may select one individual, whom he may delusionally endow with evil powers, and plot and plan for his or her destruction. And in such cases we have specially to protect such persons towards whom the patient has conceived an antipathy. Often because of some real slight or injury, sometimes from unreasoning dislike, the patient will develop an extreme vindictiveness as of an elephant or dog, supporting it with delusional arguments, and seeking an opportunity of homicide. To such cases the remarks offered as to the constitutional predisposition to suicide may be applied also to homicide, but translated into terms of the others'-life-preserving instincts. The instinct not to hurt or kill others is strong in our race, or if not always so, is carefully educated in most people.

AGITATED OR EXPRESSIVE MELANCHOLIA.—These varieties of melancholia which I have indicated are obviously a transition of the disorder downwards and outwards. The morbid state in simple melancholia is centralised. In delusion, when the mind thinks about itself and explains itself, the emotional content is diminishing; in suicide there is a distinct though insane activity of a purposeful kind; in homicide it is still less central, more outward. And in the variety called agitated melancholia the outward activities, though on a low level, are very manifest. The patient wrings her hands, tears her hair, sways up and down on her chair, or perhaps scurries about the room like a frightened rabbit, moans, groans, or cries aloud. In short the condition is one in which the gymnastic of distress in its most extravagant form has supplanted the distress. The consciousness has become a motor consciousness, and probably very little pain is felt. Actual shedding of tears is uncommon, and, moreover, when the patient is left to herself, the agitation often diminishes, and she is capable of apparently sane conduct. It is in relation to the presence of onlookers, or the knowledge that people are near, that is the occasion of agitated melancholia. In that respect the muscular activities of agitated melancholia differ from those of mania. Maniacal acts do not always express joy, are often without reference to the central state. Melancholic acts usually express grief, are the recognised medium of expression of the distressed consciousness. But angry mania and excited melancholia are not far removed.

Any attempt to interrupt these movements of grief or of fear is usually resented, and the muscular pose becomes one of resistance. The consciousness of fear begets an active distrust; or at least the conduct of a resistive melancholic seems to imply that. But we err in assuming, in the insane, a mental content appropriate to their conduct. Their consciousness probably does not contain what the muscles express, and perhaps resistiveness is merely a muscular reaction implying an unthinking aversion to interference. These resistive cases are most difficult and call for most skilful nursing. The worst of them stiffen on the very approach of doctor or nurse, cling tight to the chair if you try to raise them, root themselves to the ground if you try to make them walk, clinch their teeth if you would feed them, refuse to swallow, to wash, to dress, to undress, or even to empty bowels and bladder. Further, they often kick and bite, and scratch and pull the nurse's hair. They call for immense patience, and people are apt to regard them as wilfully disagreeable. That is because of the outwardness and apparent purposefulness of the symptoms. Even the most uninitiated recognise that the quiet melancholic is irresponsible. But the resistive melancholic seems responsible. Sometimes we may help the nurse by reminding her how a trapped animal, or a child in distress, will repel and perhaps attack those who go to its help, and that in resistive insanity the patient does not understand people. Chronic melancholia, which may persist for years, is usually of an active form and often resistive.

PHOBIA (*Partial Melancholia*).—Many cases, and some of them very interesting, without a general melancholia, suffer a morbid and persistent fear in relation to limited and specific things—knives and scissors, blood, and blood-red colour, narrow places (claustrophobia), open spaces (agoraphobia), and what not. Some of these phobias are developmental, and the common mind is normally afraid of gross darkness, loud noises, dead human bodies, reptiles, precipices, fire, deep water, and other things properly to be avoided. In the weak, either by development or after critical bodily illnesses, or in other neurasthenic states, the affection often relates to things not essentially harmful. The fear, whatever it may refer to, primarily occurs only when the patient is face to face with the object of it. But in many cases of a melancholic habit the patient dwells upon the fear, and, apart from any objective occasion, becomes distressed at the mere idea of the thing. Then very probably obsession will develop, in which the nauseous suggestion obtrudes itself repeatedly and persistently until the distracted patient breaks down under it. In any case, while the terror is upon him the patient is in great distress. And the panic which rivets his attention is associated with a characteristic muscular state, not unlike the gymnastic of acute melancholia, but with an added tremulousness and an accentuated feebleness. At the same time the pupils often dilate, the face turns pale, and the patient feels himself unable to move. In fact, the differences and resemblances, mental and bodily, between melancholia and phobia are analogous to those between unhappiness and fright. Moreover the treatment of phobia practically resolves itself into preventing its becoming melancholia,—by diminishing the general, and diverting from the particular, nervousness from which the patient suffers.

STUPOR

When insanity is described as a disease in the cerebral structures which subserve normal life, we imply that the function of nerve-cells and nerve-fibres is impaired in the sense that their excitability is diminished. Such an abeyance of functional activity becomes manifest, when it is far advanced, in changes which we can observe under the microscope, such as are described by Dr. Robertson. But, following that impairment of excitability in the cells and fibres of sane mental activity, there is usually very evident in insanity a hyperexcitability of mechanisms on a lower level, or, at least, an abnormal activity in them, and these secondary excesses constitute the positive symptoms of insanity. But stupor must be described almost entirely in negative terms. It is characterised by failures and losses rather than by excesses and extravagances. The patient, in pure stupor, cannot do anything except the most primitive things—cannot speak, cannot walk, cannot shake hands, cannot brush his hair, cannot button, and, presumably, cannot think. In other words, and to describe the disease in a sentence: Stupor is the cessation of conduct. But, before elaborating that remark, we must digress to consider exactly the system of functions which we must suppose are chiefly involved in the diminished excitability which characterises the disease.

In his description of melancholia, Bevan Lewis lays much emphasis upon the failures of the *muscular element of mind*, and indeed regards them as primary. The subject, however, is one which is perhaps more apposite to stupor. And that is in no way at variance with Bevan Lewis's position. For stupor is a secondary development of melancholia in which the depression of function, the increased neural resistance, has spread downwards and outwards towards the muscles. Indeed we might have described a stuporose variety of melancholia as leading us on to stupor. There is no case of acute melancholia that does not threaten stupor; and there is no stupor that is not, or has not been, melancholic.

The term "muscular elements" as applied to intelligence is easily understood. Based upon the muscular sense, it has its final expression in the constructive or inventive consciousness—in conceiving and designing things to be done and made. Its most elementary form is necessary to all perception. The shape, the size, the distance, the feel, the weight, the direction in which things are—all these and other qualities of external objects are perceived by muscular sensations following movements of the eye, limbs, and body. Bevan Lewis thinks that these muscular elements in the melancholic's perception of things may be impaired before and without an impairment of the other and passive elements of the senses, that the "space attributes" of external objects can only be realised by an unwonted effort, and that thereby an excessive sense of the "resistance of the environment" is suggested, "and the apparent energy and freedom of the will restricted." (We may doubt that, seeing that the elementary qualities of things are not known to the adult mind as discrete, but as compounded, and in course of dissolution, the compound mechanism of a given object into which its primal elements have been resolved will be the first to go.) Be that as it may, there can be no doubt of the importance of another form of the muscular element in intelligence—what we may call *projective activities.* These are not relations of what is commonly recognised as sensory activities, but are on the other side of mind. They are functions of what we know as psycho-motor mechanisms, and might be described as aspects of the mind in the act of moving. These are not only muscular movements, but mental movements as well—the forward activities of the intelligence such as occur in reasoning, deciding, choosing, planning. For much more of the mental cerebrum is psycho-motor than merely that part of it which contains the images of definite movements. The idea, the intention, the mental picture of any definite movement which we may project, is well down on the way to actual motor functions. But many of the more central processes are of this projective nature. For example, most people think about things, not by making use of mechanisms which represent conceptions of the things themselves, but of those which represent their names. And names are things which we speak or write, or which our eyes pick up from the printed page, or which we prick our mind's ear to listen to—all of them muscular elements. Many other mental processes we can describe in terms borrowed from the analogy of gross movements, and which suggest their projective nature. The understanding "grasps" things (Bevan Lewis) and "holds" them; we re-"collect" things as the attention casts about to "pick up" what is wanted; we "pursue" a subject, "follow" or "take up" a line of thought; our sympathies "embrace" things and people; the imagination "reaches out" to "touch" new things; we "hold fast" to convictions; etc. etc. And we speak of a "standpoint" or "point of view" where the main self, as it were, remains while the projective activities go on around it. Above all, personality, as was said, is determined by what a man conceives of himself as saying and doing—the things which he consciously projects. All these then, and others, are the psycho-motor activities, the muscular elements, which fail in melancholia, and whose failure determines the abeyance of outward consciousness.

In stupor the psycho-motor loss is at its greatest, and the systematic course of the disorder is directly outward or downwards towards the muscles. Stupor is pre-eminently a psycho-motor paralysis involving even the mechanisms of definite movements of which I have spoken. The patient cannot reason, cannot decide, cannot plan, cannot arrange in his mind to do anything, nor agree within himself as to what is best to be done, or, at least, he gives no evidence of such mental movements, for, as we know him by actual observation, he is simply a person who cannot speak, cannot use his hands, cannot walk, can hardly stand, because, apparently, he cannot conceive of these movements. And by and bye if the disease progresses, impairments on a still lower level will occur, and the patient becomes muscularly impotent, paretic, and atrophic.

By its nature a patient in stupor is prevented from revealing to us the mental content. He cannot express what he feels or thinks, or only in the most laboured fashion. This point must be insisted upon because some observers make very definite statements as to the actual condition of the stuporose mind, some of which are not verifiable. It has long been the custom, for instance, to regard stupor as a condition in which sensation is blunted. That may be so, but all that we can really say is that the patient does not react to the stimuli applied. And, from some necessarily rough experiments, I am disposed to think that, at least in some cases, there is not any gross loss of common sensation. Further, we cannot always take the subsequent version of the patient as descriptive of his content during the attack. We have no proof that memory is active during stupor. And yet we can infer a good deal with comparative assurance. We can confidently affirm, for example, that the stuporose state is essentially painful; we rarely see so pathetic an exhibition of profound distress as some convalescent stupors reveal. Both of the accepted conditions of pain are associated in stupor—a demand for response from enervated mechanisms, and pent-up activity in mechanisms not sufficiently used. Yet not in every case can the distress be observed, for even the gymnastic of pain may be lost, and the facial expression and the attitudes be those of almost pure anergia. The disease is to a large extent systematised in the highest motor mechanisms. It has no gymnastic in the proper sense, no characteristic muscular poses. The obvious thing in stuporose muscles is their absence of pose. The face, trunk, and limbs are expressionless and toneless.

SIMPLE ANERGIC STUPOR.—In this condition the patient can only be described in negative terms. It is one of the few insanities in which the obvious impairments of personality probably correspond to the actual disease. There is a profound disturbance of outward relations. The patient may, for all we know, feel an interest in affairs, but he cannot show it. He just sits still, or lies still in bed, does not speak, does not *use* his limbs, although he may move, is fed and relieved of excreta only with difficulty, does not wash or clothe himself. The appearance is oftener surfeited than emaciated; the face may be flushed; asphyxia of the extremities may occur; the muscles in general betoken profound tonelessness. In many cases the eyes retain what seem to be voluntary movements, and may searchingly wander round the room—a phenomenon which suggests considerable survival of sensation. Touch stimuli and stimulation of the special senses will usually elicit no response, but the application of mild pain is likely to elicit signs of distress. The reflexes in stupor are variable.

The condition is apt to be mistaken by friends and others for obstinacy, or what they vaguely call hysteria, and we may here note some points in diagnosis. Stupor, for example, must not be confounded with stupidity such as occurs in myxœdema, in adenoid affections, or in other cerebral “toxications,” nor with semi-comatose states induced by narcotics. The history of the case must settle the diagnosis, with special reference to rapidity of onset in anergic stupor, the existence of predisposing causes of insanity, and of such immediate causes as explain cerebral neurasthenia—exhausting illness, shock, masturbation, etc.; but there can be no mistake if the physician attends sufficiently to muscular, vaso-motor, and trophic symptoms, all of which evidence much more profound depression of function than in mere stupidity. States induced by hypnotism have a much greater resemblance to stupor, and may only be discriminated from it by circumstantial facts. In catalepsy (*q.v.*) and in hysterical anergia, on the other hand, the symptoms of low-level depression may closely resemble stupor, but the mental impairment is slighter and transient. Coma, such as may occur in traumatism, apoplexy, the post-epileptic state, etc., may also be confused with stupor unless the history of each case be studied. Then there is the interesting condition described as lethargy, which is not a real stupor, but a condition of prolonged sleep. Clarke (*Amer. Jour. of Insan.*, Oct. 1891) describes a woman who slept for seven years, then had a remission of symptoms for seven years, then fell asleep again for a spell of thirteen years. In her lethargic phase, when not asleep, she seemed quite alive to what happened, did not soil the bed, ate food slowly, but did not speak. After these momentary awakenings she just turned round and went to sleep again. The fact that she did not speak for eleven years brings the case near to stupor. She always slept deeper at night.

It is interesting to observe that stupor is the mental disease in which a mistaken diagnosis is most likely; and the reason is to be found in its primary and essential symptom, irresponsiveness. The physician should consider this fact, that stupor is *par excellence* a primary affection, characterised by its negative symptoms, as illustrating the fundamental distinction between primary (negative) and secondary (positive) signs of mental disease.

Melancholic Stupor.—This affection is much commoner than simple anergic stupor. In the latter the onset may be rapid, may indeed, after a great shock, for example, be almost immediate. Melancholic stupor is a slower development as a rule, is attended by obvious mental pain from the first, and usually follows bodily illness, or some other neurasthenic process, such as overwork, exposure to extreme heat, masturbation, or acute insanity. Concomitant with the bad spirits there is the anergia—the inability to intend and to carry out intentions. Listlessness scarcely describes it. It is a disability as well as a disinclination for outward activities. In every case the impairment spreads outwards and downwards, and becomes evident in the flaccid muscles, torpid secretions, asphyxiated limbs, soft pulse, shallow respiration, trophic depression. These develop slowly, and as they progress, mental pain increases. In such cases suicidal feelings and attempts are common, not usually of a deliberately determined kind, but of the impulsive variety. Melancholic stupor is prone to fulminating (explosive) activities. These may take almost any form—a shout, a somersault, a blow, destructive violence, homicide, suicide, or an epileptoid fit. Melancholic stupors must always be carefully watched. The mental content, so far as we know, is largely one of passivity. Many patients recall, on their recovery, things that happened during their stupor, incidents which occasioned bodily pain,—a fact which, as was said, would indicate less anæsthesia than many authorities assume. Such cases end variously—often in recovery, which is frequently preceded by a maniacal phase, a low-level ideo-motor activity, which ushers in the recovery of the projective activities of the higher cortex.

Delusional Stupor.—As Clouston argues, stupor is very rarely determined by a patient's delusion. In a few cases, perhaps, a sudden and abiding obsession (fixed idea) may explain the patient's immobility, such as that he is dead, or that the least movement may bring about some catastrophe. In the vast majority of cases, however, the delusion is the patient's apology for his stupor and not the real explanation of it. Finding himself muscle-bound he invents an account of his state—that he is made of glass, for example, or that his veins are full of mercury, that his head has dropped off, that his body belongs to some one else. Crudity is characteristic of stuporose delusions. They amply bear out what I have said, that delusions are due to too little, and not to too much thinking. Stuporose cases do not make elaborate and systematised conceptions as a rule, but become possessed of simple, fixed beliefs, as immediate and unrelated as obsessions. In the sphere of muscles the characteristic mode is that of increased resistance; and a really good case of resistive stupor is a remarkable phenomenon. Passing from the anergic state of mere flaccidity the muscles become the site of distorted activities, and, assuming attitudes appropriate to what the patient feels and believes, will not be moved from them by anything short of dangerous force. I am not aware that any one has measured the tension in such cases, but to the casual observer it would seem appropriate to describe it, not in terms of foot-pounds, but in those of horse-power. As a rule restiveness, even in stupor, is aggravated by the mere presence of other people. It differs from that of melancholia in that it is resistiveness to an interference with a stereotyped attitude. The patient perhaps sits with his hands on his knees—an attitude of delusional safety, and retains that attitude in resistance to interference even to the point of fracture; or, if allowed, will wear the skin away by pressure. A melancholic resistiveness is much more general. If you tried to change such an attitude in a melancholic, he would probably lift his hands to strike, or his feet to kick, or would clutch at things near, or would run away.

Cataleptic Stupor.—Many stupors develop cataleptoid symptoms. I need here only point out the relationships of this variety. Catalepsy in stupor is, for example, the obverse of resistance. It is much nearer a direct development of simple anergia. In the stuporose muscles there is plenty of energy, but it is lying fallow for want of centrifugal stimuli. Unable to energise himself, the patient may, instead of becoming resistive, take on an exaggerated muscular facility, and the cataleptoid state is the gymnastic of that mode of attention. In vol. ii. of this work, p. 165, reference is made to the cataleptic muscular state. In stupor we do not have the completely waxen condition so often as simply a disposition to retain induced poses. The feel of the patient's muscles gives us more of the impression that she is voluntarily retaining the attitude which we have imposed upon her; but occasionally the *flexibilitas cerea* is present in some degree. In any case the plastic muscular state is not compatible with delusions such as have been indicated, but is usually associated, so far as we can gather, with mental torpor and vacuity. When it is obvious, asphyxia of the limbs and trophic impairments are not marked. Sudden movements may occur alternately with it.

Mania.—In stupor we have a disease which, as has been said, is to be described almost entirely in negative terms. Secondary and positive symptoms—delusion, grief, muscular resistiveness, catalepsy—do occur; but the primary features are the muscular and mental immobility. In melancholia the negative condition, the absence of outward activities, is evident enough, but we designate the disease by its predominant secondary symptoms—the painful self-regard. And in mania, also, it is the secondary symptoms, the excessive low-level activities, which are most obvious. Mania is in contrast to stupor—a diarrhœa instead of a constipation of ideo-motor activities in a brain which has lost its highest and controlling functions. For the primary condition of mania is, as in melancholia, an abeyance of normal outward relations. True, the maniacal stage is characterised by a diminished subject-consciousness and by an exaggerated object-consciousness. But the object consciousness is on a low level and quite abnormal in its mode. The essential fact, then, is the abeyance of normal personality. That has to be repeated *ad nauseam* until physicians learn to have less regard to its secondary consequences; and it may be serviceable to illustrate it in connection with mania. For that purpose let us assume the patient to be one of an average mind, whose personality is concerned with the home and the earning of a livelihood as central elements in his consciousness. What happens in mania, then, is that he loses these attachments. His occupations become stale, he neglects his work, he disregards his wife and children, he destroys the peace of his home. If the attack be sudden, we do not think of it in this light, but in that case it is still more obvious that the patient has broken loose from his calling and from his home; he has dropped his career. And this failure of normal personality implies mental pain, and melancholia becomes manifest if, so to speak, the disease gives it time to develop. In many cases, however, the malady develops so rapidly that the melancholic stage is only an affair of an hour or two, and in cases of immediate mania the pain-stage seems to be as short as in a case of sudden death. In the great majority, however, careful inquiry reveals that, for some time past, the patient's spirits have been at least fitful—the sign of incipient failure in the mechanisms to which I have referred.

Passing slowly or rapidly through the stage of pain and subjective over-attention, the patient soon develops an excessive activity in ideo-motor functions on a low level. In melancholia there is in general a depression of cortical function, both high and low. In mania there is activity to excess on levels lower than the normal highest. And that bespeaks a pleasurable tone of spirits which has, moreover, its characteristic gymnastic. When the maniac is at his quietest his muscles assume the extensive mode. The wide-open eyes, the expanded mouth, the head thrown back, the shoulders open, the spread fingers, the erect carriage, the straight knees, are in strong contrast with the gymnastic of melancholia. And the exultation is intelligible; for the dynamical conditions of pleasure are fulfilled—rest of the devitalised mechanisms and full activity of the energetic—exercise of the imagination, of the special senses, and of the voluntary muscles. And even in mania the systematic nature of the disorder can generally be demonstrated. It is impossible here to expound the statement, but mania is not intrinsically incoherent. The maniac's sentences seem incoherent to us, but they are coherent to his surviving mode of consciousness. Could we obtain a complete bird's-eye view of all that happens in his cortex, we should discover that his disordered activities, even when peripherally initiated, and still more when they are centrally determined, follow along paths which are organically associated. And the big types of mania are obviously systematic developments. The following varieties are those described by Clouston. But the disease is so gross, so obvious, so fully treated of in the text-books, so impossible of private care, that we need only point out salient features and remark a few of the relationships of the affection.

Simple Mania is often not diagnosed in its early stage. There is a general exaltation of cortical function. The patient is restless, active, fanciful, pleased with himself, but often quarrelsome. This stage is characterised by great activity of the high-level functions short of corrective judgment and coherent purpose. The consciousness teems with extravagant ideas, hopes and schemes, and revels in excess of affections. Now and onwards we must note the unrestrained activity of the objective relations. Every little change in the environment suggests something to the patient's restless mind. A clever companion, who cares to lead the patient on, will elicit an apparently endless flow of conversation, quite coherent, but grossly extravagant, and may lead him into any madcap adventure—a visit to the crowned heads, or to the North Pole, the purchase of an army of elephants, or of half the coal-fields of Europe. But let the guidance of the companion be withdrawn even for a moment, and the exuberant interest of the patient will fall a prey to the next suggestion his environment offers. Facility and fickle enthusiasm are the features of this stage.

ACUTE MANIA.—In acute mania the loss of function becomes more obvious. The patient is incapable of ideas and of purposive activities, even of the insane kind, which occurs in simple mania. Consciousness now is apparently on the Rolandic level. At least the patient's activities are almost entirely sensori-motor. She—for women offer the most exuberant symptoms of acute mania—has passed gradually from active ideas and projects, and the attention is occupied wholly with sense-perceptions or with simple movement, or with both. Over-attention to sights and sounds and smells—the sensory mode of acute mania—is in my opinion less desirable and apt to become a chronic insanity with hallucinations. As a rule, however, objects perceived excite an immediate motor response. The patient sings in answer to the birds, knocks over vases, shouts the name of each thing she sees, blows her nose fiercely at a smell, poses, jumps, and gestures appropriately to the stimuli of which she is conscious. And if this sensori-motor relation obtains, such a case is more easily led back, under appropriate treatment, to orderly and purposeful activities. Other cases, again, are of a predominantly motor type, whose muscular activities seem to be subjectively determined. The outward relation is at its acme in such cases, at least quantitatively. The patient dances and gestures like a dervish to the point of exhaustion, or talks or shouts or sings incessantly. In the latter case ideation is at a minimum, and words, not sentences, pour from her lips, though she may repeat set phrases or snatches of poetry or song. Quite often she will suffer a *diarrhœa verborum,* whose only coherence is a rhythmical or rhyming or alliterative or other non-ideational association. These are the cases most likely to become exhausted, and liable to end in stupor or in acute delirious mania or in dementia.

ACUTE DELIRIOUS MANIA.—*Bell's Mania.*—In acute delirious mania the reduction of cortical function is still greater, and is accompanied by grave bodily symptoms. The mental condition is one of delirium. The patient is unconscious of things around her as such. She stares at them, feels them, picks them, as in a dream, moves restlessly about in a wandering manner, as if looking for something, or as if trying to get away even through walls and doors, and only mutters if she speaks at all, or cries feebly or whines. And meanwhile she is physically exhausted and febrile, appetite is gone, the tongue is dry and heavily furred, there are sordes about the mouth, the breath is foul with an ante-mortem odour, the pulse fast and irregular, the respiration shallow, and not proportionately rapid. Sleep is impossible. The case is now one for most careful nursing and sustaining food, which usually must be administered by the tube; and, in my opinion, rest should be obtained by mechanical restraint. This condition sometimes develops rapidly, or, less commonly, follows a prolonged acute attack. The symptoms certainly suggest, over and above exhaustion, some cerebral toxication such as we suppose to be the part cause of other febrile insanities. No one knows whether the toxines are the products of extreme nerve waste or of paradigestion, or from some other source.

DELUSIONAL MANIA.—This condition, as Clouston observes, is to be distinguished from delusional melancholia and from chronic delusional insanity, only by the fact that the patient's conduct is maniacal. Indeed there is no line of demarcation, for the delusions in these cases are often of a distressing character. Delusions abound in simple mania, but they are fleeting. Abiding delusions now referred to are the conceptions of a mind in chronic mania, such as express resentment for supposed injustice—that people have designs upon the patient's property or character, that certain people in their jealousy have conspired against him, that, while he is detained in the asylum, his enemies are wrecking his home and destroying his wife and children; and excitement and violence supervene.

CHRONIC MANIA.—Chronic mania is also an arbitrarily determined variety. It is characterised by continued persistence of subacute excitement. Some patients remain violently active on the sensori-motor level for several months; but not many constitutions can stand it. As a rule the acuteness of the activity wears off and ideation is, to a certain extent, restored. The patients then go about prone to restless and extravagant conduct, and to active and fleeting ideas. In this condition we have, *par excellence*, the insane survival of the constructive mode of consciousness. Usually demented in some degree, the chronic maniac may spend his days in fashioning curious idols which embody for him important attributes, in inventing and constructing hopelessly unnecessary appliances, or in designing and laboriously completing some handiwork which he is sure you will be glad to retain as a souvenir of his regard for you. The tone of feeling may be happy or it may be resentful—the hilarious and the angry types of acute mania as distinguished by G. M. Robertson. The imminence of dementia is usually obvious in such cases.

EPHEMERAL MANIA.—*Mania Transitoria.*—This variety is distinguished by its sudden onset and offset. It must be regarded as an epileptic equivalent. The patient suddenly suffers an abeyance of the functions of normal personality, but does not fall to the ground or lose consciousness in the ordinary sense. Instead of motor convulsions—the typical clonic spasms—the explosive activity is manifest in sensori-motor violence often destructive, such as occurs in ordinary acute mania. The duration of the attack is to be measured in hours. Then, after something like a post-convulsive coma, or sleep, the patient awakes to resume his normal activities.

RECURRENT MANIA.—To the above varieties we may add that of recurrent mania—states of subacute excitement associated with a delusional point of view, which occur periodically both in men and in women, but especially in women, and generally related to menstrual functions. These are usually chronic cases, manifesting between times a fairly sane consciousness but often slightly demented, yet subject at the menstrual period to recurrences of acute excitement. Such periodicity, common in nearly all forms of insanity, betokens a preponderance of inherited proclivity. Nothing further is to be said about these cases except as regards prognosis. Only when the patient has survived at least two periods without a maniacal relapse, may we assume that her convalescence is assured.

ALTERNATING INSANITY.—All intelligent persons alternate to some extent between phases of happiness and of unhappiness. These are modes of consciousness which respectively regard the environment as more or less at the disposal of oneself; or oneself as more or less at the mercy of the environment. Cheerfulness persists just so long as we stand up to life; apprehension darkens into sadness in the minds of those who go down on their knees before it. And these two attitudes of mind severally distinguish melancholia and mania. In melancholia the mind is reduced to a primitive level of fugitive consciousness; in mania to one of foolish activities. But in many cases—and these are always interesting—the insanity reveals a systematic and more or less regularly periodic alternation between the depressed and the exalted phase. Such cases usually, indeed nearly always, have a spell of sanity in their cycle, and that is a strange and interesting fact. The patient recovers from melancholia perhaps and seems quite well for a period; then, with a scarcely perceptible stage of depression, passes into mania for a period; then to melancholia again, and so on. And nothing so impresses upon one's mind the idea of the systems of subconsciousness as one of these cases. Here is a man beneath the surface of whose mind is an organised system of fear, beset with gloomy feelings and with a varied assortment of terrors. Each time that he becomes reduced to this fugitive level, he feels the same kind of grief, entertains the same kind of delusions, and wears the same expressions and attitudes as before with very little variation. But he has also a subconscious phase which has reference to his surroundings from the point of view apparently that the world and all its delights are entirely at his disposal—a phase of consciousness that revels in primitive and puerile activities. And again, each time he reverts to this maniacal phase, he behaves very much the same as when last he was reduced to mania—makes love in the same silly phrases, entertains the same ridiculous projects, adorns himself similarly, walks with the same jaunty gait. And as we note these phases of consciousness, their distinctive gymnastic, and their coincident and characteristic trophic reactions, we cannot but assume that, in such a patient, subconscious mechanisms are deeply organised to represent them. Moreover, the melancholia of such an one does not essentially differ from that of a patient whose disease never proceeds to mania; nor does his mania materially differ from that which never alternates with melancholia. We are forced to the conclusion then that, in the minds of all who become insane, there was a systematised melancholic or a systematised maniacal character in their subconsciousness or both. But we are probably safe in assuming that these subconscious arrangements characterise the brain of the human species, and that they would inevitably be revealed in any one who, having lost his acquired personality by disease, was reduced to a level of consciousness of an emotional form. (The same kind of things might be said of delusional types.)

Over and above the impression which alternating cases give us of the systematic nature of cerebral arrangements on levels lower than that of sane personality, we must be struck by their periodicity. To watch a man through a *folie circulaire* is to have it suggested that the human brain works in cycles, that each cycle reveals several phases of consciousness in strong relief, almost as dissociated as in the insanity of alternating personality; and that cyclism must be briefly considered before we describe the types of the disease.

PERIODICITY.—Nearly all brains that are active, and all that are constitutionally disordered, manifest a more or less regular cyclism. Nor is such periodicity a new or a strange thing. It pervades the whole of organic life. For the primary factor in its occurrence we must go back to astronomy. The same rhythm by which the stars in their courses are set, and which brings our earth's surface near the sun at definite intervals, determines also the orbit of man's brains—sets it to a measure that rises and falls with a regularity that we scarcely notice except when disorder reveals it in excess. These immense, unconscious influences are quite beyond our reckoning, but we may surmise that our terrestrial nervous systems would be grievously upset if they were suddenly transported into the rhythm which besets Mars or Venus, or still more that of Jupiter, as surely as many suffer agonies of discomfort on quitting the stillness of land to embark on a swelling sea. And though the ship's mechanical motion has a very short wave-length compared with day and night, and these again as compared with summer and winter, yet are the latter further reaching in their effects. We must think of the diurnal variations of our atmosphere as a great pulse-wave of heat and of light which determines all the nutrition of our earth's surface, and which imposes a rhythm upon all organic activity, from the production of chlorophyll to the weaving of dreams. All over the world the heart muscle and the muscles of respiration are contracting at a measure which, taken in relation to the weight of the individual, bears a constant ratio to the time of the earth's revolutions; and, over and above these regular ripples of activity, our massive movements also occur in diurnal wave-lengths. The next big rhythm is of a monthly type, both in men and in women. The lunar coincidence is the only factor which we can suggest as an explanation of that. At all events the fact is that, in nearly all women and in most men, cerebral changes occur in monthly periods. That rhythm, and the largest of all, the seasonal cycle, has an indirect influence on cerebration reflexly through the sexual and the reproductive functions. Under the influence of natural selection, organic changes in the female have become stereotyped in a rhythm which determines a rise of sex-consciousness in the spring, or, by an elaborate adjustment to the period of gestation, makes the birth of the offspring likely to correspond with the most suitable climatic conditions for its early life. From reproductive habits of a seasonal type the human race has of course departed. But the constitutional changes which are so evident in lower animals as of a seasonal nature, are the inheritance of our species also, and are organised throughout our whole nature. For the teleology of female changes applies also to the male. Animal life, in males, is largely a reaction to female states, and the organic energies of the two sexes usually wax and wane in harmony.

From the strict type, the human race is obviously diverging as civilisation diminishes the direct influence of climate upon health. A woman's concealment of menstrual symptoms is but a crude sample of a general levelling process which polite society enjoins upon men and women. Even pregnancy and lactation are considered impolite, and, with the general suppression of these and other appearances of reproduction, periodic reactions in the race are being obliterated. Equableness is now indeed a feature of cerebral stability. But in incidental insanities and in abnormal mental reactions periodicity is conspicuous. Clouston and others regard these periodic exacerbations as an almost sure sign of a neuropathic diathesis. In other words, while all constitutions are subconsciously of a rhythmical organic habit, only weak brains fail to rise above and to conceal these alternative exaltations and depressions.

PERIODIC MANIA.—The recurrent variety of mania is the first form of periodic insanity, though melancholia may also manifest regular exacerbations. As was indicated, *Recurrent Mania* is most obvious in women, because of its relation to menstruation. But it occurs also in men. Sometimes it appears that the sane period signifies an access of energy and that the mania occurs on the subsidence of vitality. More often the facts of the case suggest that sanity suffers a periodic interruption occasioned by an access of vitality in low-level mechanisms. I have at present two cases which certainly offer a contrast. One of them, neurasthenic and anæmic, suffers a periodic relapse into mania. The other, robust and buxom, seems a chronic maniac, interrupted by periodic relapses into ephemeral sanity.

CIRCULAR INSANITY.—The classical variety of cyclical insanity, however, is one which alternates between melancholia, mania, and sanity. These are cases of gross insanity specially interesting to the general practitioner because, if the rhythm has a grand wave-length, the patient may return home for months or even years, and it then becomes the practitioner's duty to watch for the relapse from sanity. And, while these cases are, on the whole, of a bad prognosis, a great deal may be done to postpone the relapse.

Theoretically, these cyclical cases might be expected to manifest a periodic reduction of personality, with melancholia as a half-way stage between sanity and mania both on the down-grade and on the upward return, thus: sanity, melancholia, mania, melancholia, sanity. But the disease is very irregular and the melancholia is often not observed, especially on the way from sanity to mania. Moreover, the periods of duration of each phase are, as a rule, irregular. The rhythm may be set to a measure of anything from a few hours to a few years. As types, however, we may discriminate three: those presumably determined by diurnal variations of vitality; those whose rhythm approximates to the monthly type; and those which are some modification of seasonal periodicity. Clouston in his text-book has a classical description of the last form. The periods were as follows:—

I. Cycle.—Mania, 6 mos.; melancholia, 3 mos.; sanity, 6 mos. = 15 mos.
II. Cycle.—Mania, 12 mos.; melancholia, 6 mos.; sanity, 6 mos. = 24 mos.
III. Cycle.—Mania, 10 mos.; melancholia, 6 mos.; sanity, 8 mos. = 24 mos.
IV. Cycle.—Mania, 13 mos.; melancholia, 6 mos.; sanity, 14 mos. = 33 mos.
V. Cycle.—Mania, 24 mos.; melancholia, 12 mos.; sanity, 15 mos. = 51 mos.
VI. Cycle.—Mania, 3 years; melancholia, 2 years; sanity, 1 year = 72 mos.

After that the patient suffered an almost fatal neuralgia, followed by some years of sanity. In all such cases it is not so likely that a particular phase will have a duration equivalent to a given seasonal or menstrual period as that the change from one phase to another will occur about the zenith or about the zero of vitality. From these grand (seasonal) variations down to the menstrual types, intermediate varieties abound. So also between these and the petit (diurnal) forms. Clouston quotes another case, "D. D." æt. 49, "who for twenty-six years was subject to regularly recurring brain-exaltation every four weeks, almost to a day." In others, the cyclism is of a fortnightly or weekly wave-length or thereabouts; and, in still others, diurnal or shorter. Many patients go to sleep in melancholia and awake to mania or to sanity, which again will be only of an ephemeral duration. Others suffer a forenoon melancholia and an afternoon mania. And in all such phases we have to note motor, sensory, delusional, and volitional concomitants. In some, stupor characterises the melancholic phase, and in others, delusion; so also the mania may be simple or acute, delusional, querulous, or hilarious.

Over and above the care of these alternating cases in their sane phases, the physician may render great service in detecting cyclism in neurotic youths, and in prescribing a mode of life which will modify it.

(It is interesting, as was said, that most of these cases come back to sanity at some arc on their cycle. It is comparatively rare for a patient to alternate periodically between melancholia and mania. Also, why do we not often find alternating cases with fixed delusions characteristic of each phase? In alternating personality (double consciousness) we have such a condition, though it is not markedly periodic. It is considered later. (It will be noted that periodic or recurrent mania and alternative personality may be variously classed. Insanities are not capable of exact and exclusive classification.)

Delusional Insanity.—There is no kind of insanity about which so much confusion prevails at this moment and so much difference of expert opinion as about the varieties now to be considered. The practitioner who tries to keep himself in touch with the literature of insanity must be in a condition of great perplexity concerning delusion, unless he is shrewd enough to appreciate the fallacies which beset asylum work.

To unravel this tangled skein will be the work of many years of careful inquiry and of clinical and pathological comparison. The following account of the subject must therefore be regarded as tentative, and it is right to say that many advanced students of delusion would regard it as quite misleading. Some fallacies, however, are so important that they must be pointed out. In the first place, most authorities describe delusional insanities chiefly from observation of asylum cases of long standing. These, it need hardly be said, are of the least practical importance, though of profound interest from a scientific point of view; and, compared with delusional states of a slighter degree, such as are met with in general practice, they are few in number. Another source of much error is that we attach far too great an importance to the refinements of foreign observers. Magnan's insanity (progressive systematised delusion) very rarely occurs in this country as a typical form. Again, we must note that very great confusion has arisen from the perverted use of two terms—paranoia and degeneracy. Any one who cares to make reference to recent English work, to say nothing of American and continental, will wonder why we have not entirely discarded the term paranoia. One could hardly conceive a greater discrepancy in scientific opinion than occurs for example in the accounts of paranoia given by Clouston (last edition), Bevan Lewis (do.), Campbell Clark, and Macpherson, in their text-books, and by Conolly Norman in Albutt's *System of Medicine.* Paranoia, however, is the subject of a special article by Macpherson (*q.v.*), and it is best that we should restrict the term in this work to the interpretation which he puts upon it. And as to degeneracy the same confusion prevails. The degenerate is, in fact, as has been said, very elusive except in his superlative degrees, and we err on the safe side if we withhold our diagnosis of him except in that form, that is, as a criminal, either in intention or in fact, a person incapable of morality and usually of a correspondingly degraded physique.

Considering that the human mind is admittedly and properly regarded as having three great aspects, thought, feeling, and will, it would be strange indeed if a great many cases of incidental insanity were not chiefly characterised by disordered thinking, without a proportionate volitional and emotional disturbance. All the same, the prime fact about intelligence is its inevitable correlation with all other aspects of mind. No one proposes, or could possibly expect, that a patient should reveal simple delusion. Bevan Lewis goes so far as to say that "monomania is evolved out of melancholia and maniacal perversions—as a special derivative of these conditions, and as one of the terminations in chronic insanity." So far as I know, he is alone in that opinion. But, in any case, monomania is descriptive of but a few delusional cases. Before a patient becomes delusional, that is, when his thinking mechanisms fail in their higher phases and become active on a lower level, some mental pain, of however short a duration, must be presumed, as it always must be in states of cerebral exhaustion. But that does not constitute melancholia. Nor do delusional cases always conform to what we have agreed upon as the secondary characteristics of mania—an excessive activity of an outward form on reduced levels. Delusion is not such an excessive activity. We cannot repeat too often that delusional cases as a rule suffer from too little, and not from too much thinking. Patients who entertain delusions, if they thought enough, would realise that they were believing nonsense. There are many cases of melancholic delusion, or delusional melancholia, just as there are others of maniacal delusion, or delusional mania, cases in which, with the essential signs of melancholia or of mania, disordered thinking is conspicuous. So also with stupor. Such cases have been referred to. But those are not the delusional insanities now to be considered. They constitute the connecting links between melancholia, stupor, and mania on the one side and simple delusion on the other. For all the types of insanity that we are considering have alloys which closely connect them one with another. Even in one case we may observe insanity manifest in melancholia, stupor, mania, and quiet delusion in successive stages.

When a patient "loses his head," that is, when the mechanisms of normal personality fail, he may be reduced to a level of painful self-regard, which is melancholia; or suffer also an abnormal resistance in the mechanisms of expression (projective), which is stupor; or become outwardly active on a lower level, which is mania; or he may suffer a rapid dissolution of all his mental functions, which is dementia. But another alternative remains of a systematic kind, which may characterise his insanity. He may be reduced to a level of consciousness in which his attention is chiefly and conspicuously, and to an excessive degree, occupied with beliefs. That is the form of insanity—a disorder of opinion—with which we have now to deal. The primary form of such insanities is a condition of confusion, in which the patient does not know what to believe. The secondary form is delusion, in which he believes the wrong thing. Both in confusion and in delusion the mental content may be one of depression, or, with grandiose delusion, it may be one of exaltation, but in neither case need there be that degree of depressed activity or of excessive activity which characterises melancholia and stupor or mania.

CONFUSIONAL INSANITY.—On this subject the unwary physician is apt to be misled by the contradictory and uncritical accounts of confusion which prevail. In particular, many observers conclude that, because a patient's statements are confusing or confounding, he is therefore confused. But the reverse is usually the case. A patient may believe several irreconcilable and even contradictory things and yet be quite sure about each. That is not a confusional state. The term *Confusional Insanity* only applies to patients who are bewildered and in doubt and hesitating. That is a state of mind which occurs as a passing phase in nearly all insanities. But it sometimes persists for a long time as simple confusion, and may resolve, so that the patient then recovers; or it may go on to delusion generally with hallucinations. The disease is analogous to *folie de doute* or swithering insanity—a condition of bewilderment and hesitation in conduct.

Confusional patients always suffer from an excess of suggestion. They cannot entertain simple beliefs, but are beset by innuendoes and corollaries. In all such cases there are cerebral activities of a form very near hallucinations and obsessions and illusions. Illusions are misinterpreted sensations; hallucinations are sensations or perceptions automatically occasioned, that is, without an apparent objective stimulus; obsession is an idea which occurs again and again, and obtrudes itself irresistibly upon the attention; delusions are mistaken beliefs, whether referring to perceptions or to oneself, or to things in general. But in insane processes there are no hard and fast lines between these various morbid activities, and the merging of one into another is clearly apparent in confusional cases. Ideational activity of an unrelated form—the occurrence of words and meanings in the mind without a proper content—are due to morbid processes in mental areas exactly comparable to hallucinations such as voices, bells, singing, and visions, which presumably arise near the Rolandic areas or below them. A gentleman who had been working hard in a hot climate told me lately that, on several occasions when resting, words, not in any recognisable voice, and having no apparent coherence, crept upon his attention in a string, such as "Mine—hut—sweet—contract—lie—," like a telegram in cypher of which he did not possess the code. The moment he turned his attention full on the process the representation ceased. If he had attributed these words to some voice, we would call them hallucinations of hearing; if he accepted them as significant and believed nonsense because of them, he would be suffering delusion. And in confusional insanity, without hearing actual words, meanings, ideas, and suggestions occur to the patient's mind and have to be explained. In particular, confusional cases suffer from a morbid process very analogous to illusion in that they lay an emphasis on certain phrases that they hear that was never intended, and ponder over them with care and perplexity. Some confusional cases are insanely mystical. The patient begins to doubt self-evident facts, cannot accept the material limitations imposed upon our senses, and constantly seeks a mystical and subtle explanation of the most simple things. These metaphysical disorders usually have a religious reference. Such patients are often slow to accept a dogmatic delusion, they simply regard everything as an open question; but such a state of mind is quite unfit for society, and it is on the way to gross delusion if not corrected. A patient, for example, who wonders whether some one else can occupy his mind, and who occasionally supposes that the things he is saying and doing may not be his own, is very near to delusion. Many confused patients are haunted by that suggestion of an indwelling spirit. I have voluminous notes written by one of Dr. Clouston's patients of this class—essays upon such problems as that of the "existence of the real objective world, which exists, I suppose, objectively and subjectively, to God and in God." His mind was of extraordinary quickness and subtlety, his nature very gentle and generous and affectionate. I often think of him as one whose mental disease is to be explained on the hypothesis suggested in a previous paragraph—that the organs of his personality were a good deal too much in advance of the standards of our time. He suffered from an alternating disorder of the intelligence. We called him well when we had induced him not to think, but to engage his muscles. When he took to thinking he usually became painfully confused and bewildered. Sometimes he then went back to a muscular life, in which he found relief; at other times he gave himself over to some expansive truth such as that "God is love," that "all thought is one," that "persons are inseparate," etc., and, revelling in the developments of such an idea, he suffered what was really a mild ecstatic mania for a few days. In that phase he could hardly be said to think. Everything was clear to him; he knew all that he cared to know; he did not require to consider. Most confusional cases, however, are perplexed by much less subtle imaginings and occupied by much more mundane suggestions. Two cases under my care, one a man, the other a woman, were very far from occultism. They were perplexed usually about quite trivial matters—wondered if somebody meant anything personal when he said that the day was overcast; or if that remark at table, "Make yourself at home," could signify a good deal more than appeared; and so on. A great many patients from overstrain or shock go no further than simple bewilderment. They complain that they cannot understand things as formerly, that in reading or conversation some irrelevant suggestion crops up and confuses them, and that their memory is precarious. If such patients can be taken in time, compelled to rest, and not allowed to think, recovery often follows quickly. As soon as the patient begins to ponder over these cross-suggestions he is in risk of becoming delusional, or if, without entertaining these fleeting ideas, he allows himself to mope over the facts of his case, he is apt to become melancholic. The primary lesion in this as in swithering insanity seems to be a destruction of common sense. That characterises all delusional states.

Systematic Delusions not Progressive.—Patients who entertain absurd suggestions for any length of time usually end in gross delusion. Their mistaken beliefs are systematic in the sense that they are elaborate conceptions, supported by a mass of supposed evidence, and they are systematically expressed in conversation and in conduct. But in many cases, though perhaps not in most—which is doubtful—they are not progressive. That is, ill-health and cerebral neurasthenia, characterised by doubt and by delusion, may be recovered from as other insanities are. Under appropriate treatment the delusion obviously loses hold upon the patient's mind; prevented from expressing it, he lets it wait; induced to occupy himself with other things, he loses interest in it; for some time, perhaps, he still thinks it may have been true, then finally he regards it as folly. These episodic delusions are much more important than progressive delusions, because of their frequency and because of their curability. The topics of them are innumerable, and we can only glance at the more common. Nor need we pause to remark the coincident effect upon the spirits which such delusions have—sometimes depressing, sometimes exalting—nor their effect upon conduct, for these can be inferred with approximate accuracy.

Delusions of bodily illness are very common, and the patient, though not the subject of melancholia, entertains the kind of beliefs which have been indicated as common in the visceral form of that disease.

Delusions of seduction often occur in women at or about the climacteric, and in girls at or about puberty. Such cases may assume a medico-legal importance. Short of these, many aggrieved spinsters come to believe that they are the victims of some one who has wrecked their affections and broken a promise of marriage. There is generally some circumstantial foundation for such delusions. So also with *delusions of pregnancy*, which are most apt to occur in unmarried women who have cause to be afraid that they are with child, and in married persons who are afraid that they are not. Of a similar kind are the delusions with which we are familiar in hysteria and which refer to a supposed *loss of power*. The patient believes, perhaps, that she cannot walk or cannot hear properly, that memory is failing, that she is entering on her dotage or becoming insane. These mistaken beliefs may occur independently of a hysterical condition or persist beyond it. All such delusions referring to bodily states are occasioned in part by peripheral or systematic stimuli, but the misinterpretation of them is determined by central disorders.

Religious delusions are of another class. The patient believes, perhaps, that he has a special mission conferred upon him by the Almighty by immediate revelation, or that a universal crisis is at hand, or that God is personally displeased with some one of his paltry misdemeanours, and threatens some disproportionately dire revenge. Others may be described as *social delusions* in belief of which the patient supposes, for example, that he is on the verge of making a great fortune or of losing it, or that it transpires that he is after all of noble birth, or that he is an outcast, and that society is sending him to Coventry. Then there is that strange condition to which both men and women are liable who believe or pretend to believe that they have been attacked by robbers or burglars, or fired at, or poisoned. All these general delusions are to be attributed to central rather than to peripheral disorders.

MONOMANIA (*Fixed and Progressive Delusions*).—Dr. Macpherson describes paranoia in a special article (*q.v.*) and includes two clinical types. One of these is the systematised progressive delusion (Magnan), characterised by absence of inherited or previous insanity, occurring, that is, in strong-minded persons with a stage of inception, one of persecution with hallucinations, one of ambition or grandeur, and one of dementia. The other is the paranoia of the degenerate—a form of delusional insanity which may simulate Magnan's type, but which lacks its constancy of feature and its tenacity.

Some would have it that no more need be said about fixed and progressive delusions, but a host of accurate observers in this country recognise that the above two classes still exclude a great number (perhaps the majority) of fixed British delusions. The distinctive feature of the above two types, it will be noted, are, in the former, absence of recognisable predisposition and remarkable constancy and tenacity in the progress of the delusion; and in the latter an obvious diathesis and much variability in the progress of the delusion. Exceptions in both respects abound. There are, on the one hand, many apparently strong-minded persons, not obviously of the insane diathesis, who become the victims of a systematic and progressive delusion which does not even approach to Magnan's type in its course. Some of these are delusions of exaltation not preceded by a stage of systematic persecution. Others are cases of delusions of persecution who die of a ripe old age, perhaps softened and not so angry, but still showing no signs of passing into an exalted stage, or who obviously miss it out and become demented before they die. And, on the other hand, there are still a larger number of exceptions—cases with well-marked predisposition to insanity, whose delusions are very systematic and very tenacious. These cases, as was said, are all unimportant except from a scientific point of view. You cannot do much in the way of treatment for the King of all the Continents, and the longer he reigns the less likely is he to abdicate. We shall therefore merely indicate the nature of these insanities, and in so doing shall use the old and much-abused term, monomania.

Monomania of Persecution.—A systematised and progressive delusion of persecution often arises after an attack of mania or of melancholia, or it may be occasioned by bodily disease or other cause of malnutrition, or it may develop gradually in a suspicious mind. Reverting to what has been hinted in considering Alternating Insanity and previously, we have to regard delusion of persecution as an expression of the fugitive consciousness in the sphere of ideas, as melancholia expresses it emotionally. The patient gets to feel that he is at the mercy of his environment and that he is being worsted. Querulousness and grievance-hunting, as in Magnan's insanity, characterise the inception of the disease, and, by degrees, insane suspicion ripens into gross delusion and persists. From an early stage the disposition to believe evil is supported by peripheral processes, by paræsthesias, or by hallucinations. The exact expression which the patient finds for his sense of persecution matters very little. But we have to note two directions in one of which nearly every monomaniac of persecution seeks his explanation. Some patients believe themselves to be the victims of a conspiracy of known people, or of one or two people. Others refer their supposed evil plight to unknown and unknowable agencies—mesmerism, electricity, evil spirits, or drugs. When the patient insists upon the bodily evidences of his persecution we may be sure that he has some real trouble to complain of. Take this letter, for instance, quoted by Clouston—a good sample of such complaints: "Forced dreaming, forced vomiting from the stomach, forced glut vomiting from the throat, cold shivering by the forced thinking, sweating done in the same way, pains in the stomach any way they think. I think it is time that this way of punishment should be stopped. . . ." These "punishments" are obviously bodily stimuli. And the quotation, in the recurrence of the word "forced," illustrates a not uncommon symptom, the use by the patient of a kind of slang (neologism) which he invents or adapts to describe his sensations. I have a patient now whom it is impossible to understand until you have learnt his vocabulary. He believes himself persecuted by what he calls "Thugs." But they are not Thugs as we know them but evil spirits who haunt the air, and who can even come at him through the earth, the trees, or walls. "Doctor," he writes, "I am at my last point. Such work these people are getting in on me as you can't possibly know. They have taken the outer circle, and they are verging more and more, prising it for all it's worth and far more than is legal." So also in cases cited by Campbell Clark in discussing Insanity of Persecution—"That I have got attraction to my frame, medical attraction to the atmosphere; that they are taking me in from Glasgow, doing as I am and has done; that they have controlled me to mechanism, steam or wind, gauging of the atmosphere; that I have controlled my frame to his caturging or rifle pits in Lenzie" (from a letter by a male patient). A female patient uses this same word "frame." "They took in my frame in concealment with their weapons." "They cannot telephone without a female frame being taken in." "I didn't come to expound the weapons and Mr. Tennant will let out my frame." These neologisms are of interest as suggesting feelings and ideas which no known words are sufficient to describe. And when a patient has these quaint paræsthesias, or when hallucinations occur in explicit support of the delusion, recovery is not to be expected.

MONOMANIA OF EXALTATION. — The incidence of fixed delusion of grandeur may be as in monomania of persecution. There is a striking difference in the progress of the disease which has not received sufficient attention. When patients conceive delusions of persecution they do not, as a rule, change their identity. A few cases become Cain or Ishmael, the Scarlet Woman or some other unfortunate. But in the great majority it is still, say, John Smith or Eliza Jones who is suffering persecution. In connection with delusions of grandeur on the other hand, patients almost invariably assume a changed identity. They may become a royal duke or Jesus Christ, the Empress of India or the Wife of God, as if to account for the good fortune which they are persuaded has befallen them. But they almost never believe in a delusional good fortune befalling John Smith or Eliza Jones. When a patient conceives a delusion of good he almost invariably changes his identity and assumes a new title: those who conceive delusions of evil, retain their own identity, but assume that other people have taken on a new character. For a difference so notable, the only explanation that offers itself is the very obvious one that it is a predisposition of all human nature to throw the blame of a misfortune upon one's neighbours and to attribute to oneself all that is of good.

Another point requiring explanation is as to the cause of a sense of well-being in disease. We can understand the patient who, in failing health, feels bad all over and takes a gloomy view of everything. But how does disease occasion a sense of good fortune? In phthisis, in general paralysis, in alcoholism, a delusive hopefulness is often to be noted, and a similar anomaly presents itself in simple mania and in delusion of grandeur. In the latter instance the patient is on the rapid way to dementia in all probability, and yet he feels grand. The explanation of all such feelings probably is that, in a brain so diseased, the physical conditions of pleasurable feeling prevail. These are, as has been said, a series of reactions in mechanisms which are not called upon to discharge more energy than they can afford. In simple mania, in general paralysis, and in delusion of grandeur, the exhausted mechanisms are at rest. The patient does not even stimulate them by self-examination. And the lower, unimpaired centres are active—especially those concerned with actions and those which subserve imagination. That would imply that the melancholic brain, and the brain which suffers delusion of persecution, is less deeply disordered than the brain of mania or the brain which conceives delusions of grandeur. The latter have lost nearly all self-consciousness and are the seat of a low-level activity. They have dropped their exhausted personality. The clinical facts of these grandiose cases need not be elaborately reviewed. Filled with a sense of good fortune, the patient believes that he is a person of great rank or of great wealth. As always, his conduct does not really square with his delusion. In particular he does not demand with consistent dignity that other people should acquiesce in his belief. His mind, so reduced as to believe the preposterous about himself, is demented enough to be facile and easy-going. Nearly all his expression of his delusion is in the sphere of appearances—in dress and in manner. Believing himself to be heir to the crown, he may assume a pompous air and may don a royal dress. But he smiles if you treat him as a lunatic, his crown is probably of straw and his royal robe a tawdry thing in tatters, he never concerns himself with the serious affairs of state. And the same superficiality and insane insincerity characterises patients who believe, for example, that they are Christ, Shakespeare, Crœsus, or Sandow. All such cases, when the patient assumes a new character, the personification of some kind of grandeur, and abides by it for years, are likely to become demented or to die in their delusion. Such insanities signify a reduction to a level of consciousness which is the converse of the fugitive—a refined mode of the predatory state of mind; an ambitiousness which regards all that the world holds of good as rightfully its own.

ALTERNATIVE PERSONALITY—(*Double Consciousness*).—Cases of alternating personality are endlessly interesting and suggestive. And their special attraction, like that of some other mental disorders, seems to be because they exhibit in exaggerated form modes of consciousness with which we are familiar in sane life. We find it useful to take the simple mind as our normal standard and select its prevailing interest, say the domestic, as determining personality. But the more active human minds are far too complex to admit of such description. Such persons especially as are aware of a conflict between what they call their better selves and other phases of themselves which they do not understand and cannot manage, can at least be said to have interests and pursuits which are without their personality. Indeed we must note a type of persons whose different phases seem hopelessly discrepant and are in fact not at all bound by any unifying process in their consciousness. Such people can be said to have two or more personalities—as difficult a metaphysical conception as that of the Trinity. And this dissociation of the various phases of a mental life, though it has not received much attention from alienists, is a very important factor in insanity, even when there is no obvious question of morals involved. The dislocation which such minds suffer is metaphorically described by Robert Louis Stevenson in his "Strange Case of Dr. Jekyll and Mr. Hyde" as a profound bodily disturbance. But even when there is no such conscious antagonism between the two or more personalities, there must be a cerebral dislocation, a dissociation of two or more great kinds of activity, and such brains, whose parts are not subordinate to a central function, are certainly precarious. This subject is too large for our summary view of insanity; but it is too important to be passed over; and a glance at some aspects of it which seem important in practice may suggest others which cannot be remarked upon.

To appreciate the significance of alternative personalities, we must understand something about subconsciousness. In particular, we must note that, though the mind is not conscious of it, the brain may carry out a synthetic, a constructive process. That happens daily. The child, for example, acquires the elements of things—the yellowness, the softness, the smell, the shape of an orange, let us say—as separate; but his brain compounds them into a whole. And every one of active mind knows how things resolve themselves in sleep. You go to bed with two and two in your head, so to speak, and in the morning they have become four. The same thing happens when the mind entirely leaves a subject for a while and returns to it again. In the interval the subject has developed: that is, subconsciousness has been busy with it and the salient features of the thing stand out clearer than before. A man, for instance, who spends his days in the city and returns each evening to music, finds that, while his mind has been busy in the city, his music-brain has been making progress; and next day again it appears that, while his mind had been wrapt in music, his city-brain had solved some of his business difficulties. But these developments may not be all pleasant, and they may occur in relation to things which the mind has not attended to for years. The revival of long-past images in a new and compound form is common in primitive intelligences. By some such process we must probably explain the adoption by the insane of an entirely unexpected personality. The young mind perhaps was impressed by some Bible character or by some dramatic personation, and, years after, when normal personality subsides, the patient awakes to the fact that he is the prophet Daniel, or a brother of Prince Charlie, or Solomon himself, or perhaps King Lear. And when that happens, the patient does not have to think how such an one would feel and what he would say or do. The brain has made a character-study for him and already knows the part. Far more commonly, however, the character to which the reduced consciousness attaches is one familiar, at least in its essentials, to the mind. It is a vice, for example, of hosts of idle minds to revel in day-dreams of an attractive form. Factory hands, we are told, are much addicted to the dissipation of resorting to a fancy-self, a glorified self, who perhaps lives in a castle, with many horses and untold servants, or who has wardrobefuls of fine dresses, countless admirers, and unlimited opportunity of romantic adventure; and those who have studied the mental habits of that class tell us that these people revert to this fancy-self when they have nothing else to think about, and, day after day, imagine themselves in their romantic character, enjoying a fancy-life in ever varying circumstances. If these things be true, we have here an explanation of the assumption by the brain, when normal personality wanes, of a grandiose mode of consciousness and of a name which expresses it. For historical characters, long since dead, are not the most popular in insanity. The most commonly assumed identities are living celebrities and their relations, and nearly always aristocratic celebrities whose names are an emblem of rank and power. Many persons who pass for religious are victims of a like kind of vice. Their religious phase does not bind their whole life together, but is dissociated from general activities and is often almost entirely sensuous. It is a phase to which men and women resort when they wish "to forget the world," and then they languish in florid imagination and in unbridled feeling, very much as if under the influence of a narcotic. Such dissociations perhaps explain some of the religiose forms of insanity. Compare also the love-dreams of some minds, fed on sentimental novelettes; also the adventure-dreams of message-boys who gorge themselves on "penny dreadfuls."

Still more intelligible are those cases of alternative consciousness of which the two phases correspond to two actual stages in the life-history of the patient. Last year such a case came within my knowledge—a German colonel who spent his latter days in England. After an apoplexy he became aphasic and forgetful, subject, as these paralytic cases often are, to emotional excesses, and continually semi-delirious. Some days he was in a German phase, speaking German nearly all the time, and recalling German people and incidents. Other days he spoke English almost exclusively, and his reminiscences referred chiefly to English experiences. In his less pronounced phases, his disordered consciousness embraced both German and English images. Of such varieties, L. Bruce's[1] case is a good sample. His patient appeared to alternate between a right-sided and a left-sided celebration, with states also in which both hemispheres were active. At all events the man was in one phase an alert, mischievous maniac, whose sensibilities were keen, who spoke English, used the right hand freely and wrote, not very clearly, from left to right; and in another phase he was demented and dull, manifested unmistakable impairments of sensibility, spoke a kind of Welsh gibberish, carried his right hand usually in his pocket, but used his left freely, and wrote with his left hand in mirror-fashion. He also had ambidextrous phases whose mental state was a mixture of the English and Welsh modes. But the dissociation was so complete that in his pronounced Welsh phase he entirely forgot the facts of his English phase, and *vice versa*, though his English consciousness retained facts of its own phase.

But there are still other cases which cannot be explained as the revival of actual stages of a life's history. Many cases have been recorded of alternation between the real personality and that of some one else. All that we can surmise about such cases is that the brain had received vivid impressions of that other self, had subconsciously compounded them, and assumed the new personality at times and for a brief period to revert again, after a sleep perhaps, to the modes of activity of the real self. And, short of the assumption of a new identity, the loss of the real identity is very common in disordered minds. Many patients complain that they feel they are not themselves, that when they speak it is some one else's voice and not even their own opinions which we hear, or that the things they do are not their own actions but those of an interloper.

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[1] *Brain*, vol. lxix.

All such impairments of normal identity are peculiarly liable to occur in persons whose activities are too often dissociated. Stable brains are brains in which one mode of consciousness prevails and to which all other activities are subordinate. A brain which entertains diverse interests and pursuits not correlated by any unifying idea or feeling is by nature unstable, and an abrupt failure is apt to occur when one phase of the mind includes a conscience which will not condone the pursuits followed in the other phase. Tandem-driving is a precarious mode of progression when the tracer wants to be leader and the leader disapproves.

Insane Defects of Inhibition.—Sane inhibition is obviously impaired in all acute insanities. Melancholia or mania supervenes only when normal personality fails, and that implies that the patient's sane will is lost, though he may manifest determination or impulsiveness in disorderly directions. In Stupor, as we have seen, the expressional functions are profoundly disordered, the muscular or projective elements in life suffer great impairment, and anergia prevails. It may be that the will in its more central phases remains; that the patient is *trying* to behave sanely though the resistance in the ideo-motor and Rolandic mechanisms offers an insuperable barrier to the transmission of his dictates. We cannot say. But there are conditions in which inhibition is primarily impaired without any great interference with the rest of the mind such as we can appreciate. The best cases in point are those characterised by *Aboulia,* which is a simple anergia not usually so gross as to demand asylum treatment. The patient in the first instance simply fails to do the things which he has intended to do. That phase is common in a great many minor disorders, notably in those of alcoholic origin. But a deeper reduction of volition occurs in true Aboulia, when the patient suffers a loss of energy sufficient even for automatic acts and habits. And it is at the point of change from one kind of activity to another that the failure occurs. The affection is one which does not allow of interruption of the actual mode of consciousness of the moment. In the morning, when the patient ought to get up, he just stays on in bed; at bedtime he does not undress; he sits at table and cannot begin to eat; out for a walk he cannot turn to come home; he will sit for hours and not offer a remark. And all the time the mind is not profoundly affected. The patient is aware of his disability, and can, when once he starts, converse freely about it. But the risk is that, if rigorous treatment is not adopted —enforced regularity of hours, enforced hygiene, and enforced distraction from introspectiveness—the patient will become further disabled. Aboulia may go on to stupor—a systematic progress of the disease outwards and downwards towards the muscles. Or it may take a more central course and develop into *Folie de doute* or Swithering Insanity. Then the patient is liable at any moment, however inopportune, to stop and wonder whether he ought to go on. That is, his intelligence is becoming involved, and a vicious habit is established in which the patient fails to do things, wonders why, wonders how she can, wonders whether she should, and still does not do them. This condition is, in a sense, a confusional insanity; but it is a confusion of impulses rather than of ideas. As in ideational confusion, the conspicuous feature is an excess of secondary suggestions. Its symbol should be a mark of interrogation. The patient can take nothing for granted, but questions the propriety or the advisability or the cause or the result of everything she is about to say or to do. As was said, the primary fact in such cases is the loss of common sense.

A distinctive feature of this insane indecision is to be found in the subjects to which it refers. It is not about important affairs—not about proposals of marriage, or a declaration of war, the offer of an appointment, or the investment of a fortune. In swithering insanity the patient hesitates as to which stocking to put on first, which garter, what boots to wear, which hat, whether to put the left or the right foot on the first of a flight of steps, whether to step on the lines of a pavement or to avoid them, and as to a hundred other things which likely do not matter a straw. In other words, the patient is beset by trivial questions which should have remained entirely beneath her notice. All the petty things of life should occur subconsciously, but in swithering insanity the trifles of subconsciousness confront the patient at every step. The condition is not without resemblance to Abasia Astasia. Let the physician try for a quarter of an hour during a walk to attend entirely to the art of walking—how one leg is to pass the other, exactly where each foot should fall, etc.—and he will understand how subversive of sanity it is to fetch up from subconsciousness the multitude of things that ought to lie there.

Or again, in impairments of inhibition, the mode of consciousness may be the obverse of *Folie de doute*, and the patient manifest an insane *Rhythmism* or automatism. Rhythmism rarely occurs conspicuously except as a concomitant of other and grave disorders. Its characteristic is that the brain continues to repeat a given movement indefinitely. Many patients, for example, go on repeating a single word or phrase indefinitely (verbigeration) without content or significance; or, if you ask them several questions, will give the same answer to them all. Or Rhythmism may manifest itself in what in the sane would be called habit-spasms, which have so interesting a relation with chorea. Stereotyped recurrent grimaces may occur, or the patient may go through a gesture again and again. More commonly, insane poses and attitudes and gaits insist upon constant reiteration, of which the most common, and a very unpleasant one, is a swaying motion of the trunk and head backwards and forwards. Kahlbaum described as an entity a series of symptoms to which he gave the name Katatonia, in which rhythmism and cataleptoid signs were prominent, but the condition is not now regarded as a specific or systematic affection. (Rhythmism, the repetitive mode of activity, characterises many of the insane. Insanity tends to habit. Note also the relationship of resistiveness—a revulsion against interference with the attitude or occupation of the moment, and of aboulia—an inability to stop doing one thing and to proceed to the next. Rhythmism also characterises obsessions.)

Insane Impulsiveness.—The affections previously described are essentially significant of a reduction of inhibition—a negative lesion. In others, however, though inhibition is often impaired, we have to suppose some positive activities of an excessive kind, some hypersensibility to specified forms of stimulation. Such excesses of reaction are common in chronic insanities, and it is interesting to note that these are often reversions to childish proclivities—impulses to break glass, to throw down walls, to scratch smooth surfaces, to tear, and break and spoil things. Many of the insane cannot see anything worth destroying without an impulsion to destroy it. Biologically considered, such destructiveness is difficult to explain except as a form of mischievous playfulness which becomes manifest when disease has obliterated the acquired respect for property. Less harmful and more amusing is the temptation to touch and handle everything that catches the eye, to which some of the insane are prone. Either in sensory mania or in chronic insanity the patient may reach out to touch everything he passes that is bright or of an attractive form. It is impossible to keep up a flower garden to which the insane are admitted.

A far more important class of impulses, however, is that which includes insane proclivities to crimes and to vices. There cannot be any doubt that immorality is often determined by a morbid sensibility to vicious indulgences — a constitutional excess of temptation,—though we need not, on that account, condone all such failings or expect that they will be "cured" by measures which are milder and which provide less of a deterrent against vice and less of an incentive to virtue than such measures as we adopt with sane offenders. For the understanding of these insane temptations the physician may contrast them with the phobias, some of which we have considered. In the case of these, the patient suffers an abnormal abhorrence or dread of certain things, while in impulsiveness the balance of feeling is all the other way. The subjects of phobias are not obviously the subjects of insane impulses, but we might compile a considerable list to emphasise the antithesis. Thus—pyrophobia (fear of fire) and pyromania (the impulse to set afire); gynephobia and anthrophobia or nymphophobia (sexual fear) and nymphomania; dipsophobia (fanatic abstinence, cf. hydrophobia) and dipsomania; kleptophobia and kleptomania; agoraphobia and agoramania (the impulse to take to the open fields); claustrophobia and claustromania (the impulse to take to human burrows); acro- or batophobia (the fear of high places) and acro- or batomania (impulse to precipitation).

Violent impulses often beset the degenerate, and, as was said, we cannot call a man degenerate who is of sound moral nature. Degenerates are often moral imbeciles. That is to say, they fail to appreciate goodness, to understand the value of it, even to recognise it when it is exhibited to them, and still more are incapable of being good. Over and above that general immorality they often experience a violent impulse to commit ordinary crimes, and sometimes are impelled to commit horrors over which they gloat. It then becomes a medico-legal point to determine whether the impulse, with or without other symptoms, constituted an insanity. Short of such cases as come into the police court, however, the physician often has to determine the sanity or insanity of a private patient who has been guilty of some misdemeanour. He can only do so by a systematic investigation of the patient's mental functions, including the moral sense, by an inquiry into the heredity of the case, and by noting such physical facts as might betoken or occasion a debased or unstable character. Further, the circumstances of the case must be carefully weighed; for we must remember that in many cases the patient has been subjected to what might be a great temptation even to a sane mind.

Impulses of nearly every kind are common in the insane. We may here advert to the danger of *mutilations* which mostly occur in melancholia, in paranoia, and in general paralysis. Sometimes a patient feels impelled to mutilate a neighbour, sometimes himself. Three cases occur to me, all of them patients who were living at home. One of them, who subsequently developed a climacteric melancholia, was beset by the impulse to amputate the penis of her paralytic husband, but refrained, and was sent to an asylum. The second, a puerperal case, had to leave home because of the dread of mutilating her child; and that is a common kind of danger in all puerperal insanity. The third, a lactational melancholic, tried to cut off her child's hands with a table-knife.

Many patients pull their hair out, or try to gouge out their eyes, or lacerate their flesh, or amputate a finger. Attempts at castration or at amputation of the penis are very common in men who entertain sexual delusions or who are greatly tormented by sexual impulses, and often signify masturbation. These self-mutilations are very unintelligible from the point of view of evolution. We have to remember, however, that very few people are content to leave their bodies as nature made them, and that barbaric people resorted to many devices to alter their shapes; also that mutilations, especially of sex-organs, were associated with many unenlightened religions.

In the insane, impulses are very often the result of *obsession*—an insane mental process which is closely related to delusions and hallucinations, but which is best regarded in association with impulse. An obsession is a persistent and recurring idea which forces itself on the mind and will not be denied. Obsessions cannot always be distinguished from hallucinations. The latter are automatic activities in some part of the sensory tracts which spring an unreal perception on mind. Obsessions are also automatic activities. They do not require any external stimulus, though, as is the case with hallucinations, they may be determined by circumstances. A sense of silence, of loneliness, far-off, faint sounds, or sights or smells may occasion them. Both hallucinations and obsessions occur most seldom when the mind is most stimulated by normal sensorial activities. Their prime character, then, is that they are in some degree fulminating or impulsive. They are sensory or ideational activities which correspond more or less to epileptiform motor symptoms. In mania, all manner of suggestions are sprung upon the mind. In obsession it is the same idea, again and again and again. There is another parallelism, then, between an obsession (*sensori-ideational*) and say verbigeration (word repetition, *motor*). Both are rhythmic or repetitive. Hallucinations of hearing are not obsessions, but *a hallucination*, such as that of the words "You are a fool," might become an obsession. We limit the term to ideas. Any recurrent hallucination which conveys an idea may become an obsession. But not always, for, if the patient hardly attended to the words, there would be no obsession. That is the last quality of obsession—that it invades the personality. The patient accepts the idea as his, recognises it as part of his mind. Obsessions, then, are ideas which are occasioned by automatic cerebral activities, which are repeated with great frequency, and which invade the personality. And there is no end to the variety of them. Many of them are so silly and so irrelevant that we cannot understand their obtaining a foothold in the mind. Others come very near home, so to speak, have an intimate personal reference. But they defy any attempt at classification, for, as Macpherson has it, "there are as many forms of obsession as there are of thought." For the practical physician, however, it is useful to note (1) those which are, in a technical sense, indifferent, and which only bother the patient by their persistence; (2) those which excite well-marked emotion, and in these the emotion is usually grief or fear (cf. the phobias); and (3) those which suggest some specific line of conduct and tend to become impulses. Of the last, the obsession which besets the true dipsomaniac and the transition into the corresponding impulse is specially considered elsewhere (see Dipsomania) and may be taken as exemplifying the class.

Insane impulses, whether preceded by obsession or not, are too manifold to be considered in detail. An impulse, in the technical sense, has these characteristics:—That it does not require objective stimuli though it may have an appropriate occasion; that it is persistent (rhythmic); that the patient is impotent to resist it; that satiety follows upon the indulgence of it. The most important perhaps are homicidal or suicidal. Patients who run amuck are often the victims of impulse—feel an irresistible craving to kill people. Sometimes suicide is an almost unrelated impulse, occurring in patients who are not melancholic and not delusional. Dipsomania, kleptomania, nymphomania, pyromania, are other important forms of impulse. As every one knows, these impulses may occur without any other obvious disorder of the mind. But anything that can enter into the mind of man as a thing to say or to do may become an impulse in the strict sense. A patient may be beset by the impulse to count, to turn the hands of clocks, to unscrew all screws, to look for pins, to put things in the fire, to pour out water, etc. etc. etc.

The diseases of the will which appeal most to the public are, of course, those which result in vice or in crime. Drunkenness is treated of in the article "Alcoholism," and dipsomania is specially described. That separateness of treatment illustrates two aspects of all insane vice. There is the insane impulse, as in dipsomania, due to defective organisation and the development of an excessive susceptibility; and there is education or habit, as in drunkenness, which fosters the original sin. In *sexual perversion* and in *sexual excess* both these factors play a part. In sodomy, in masturbation, and in other such vices, we have often to note a defective organisation of a negative form, comparable to a phobia. Femininism in men, masculinism in women, denote a constitutional aversion to normal love, as well as a perverted desire for the abnormal. Much more important, on the whole, for it is now very common, especially in women, is a simple absence of sexual enjoyment. It is a serious question whether modern culture implies a failure of instincts and capacities essential to the race. Still more does the importance of the subject appear when we realise how often a sexual distaste spoils the happy fellowship of man and wife, and eventually precludes the possibility of a successful home. The fact of such a difficulty is revealed so often in the consulting-room that one is compelled to regard it as a large factor in the troubles of present-day society. The defects of a too prudish upbringing are in some measure to blame for the vice. Both sexual perverts and married inverts would to some extent be prevented if youths grew up in the belief that sexual desires and feelings were not at all improper, much less wicked, but both necessary and wholesome. In proper form and in appropriate circumstances sexual activities are as essential as are eating and drinking—which is a commonplace that is implicitly denied in our present-day upbringing. Excepting in those who will not, or should not, marry, it has become a duty to develop connubial, maternal, and paternal capacity. For the rest society suffers perhaps from a too lenient view of sexual offences. There are very few cases in which punishment of them would not be beneficial. But a wise view of sexual offences is not to be insisted on until wise views upon inoffensive sexual functions have been learned.

Sometimes a task scarcely less delicate falls to the physician who is asked to determine the nature of a supposed case of kleptomania. He will in any case have to choose between describing the patient as dishonest or as insane. The culprit might usually be left to say; for most cases are both. Most kleptomaniacs are women, perhaps for want of business education. The abeyance of sufficient motive, some weak-mindedness, and an approach to the insane diathesis, are corroborative. In pure kleptomania there is well-marked impulse, but a large number of spurious cases are rather the subjects of doubtful habit—of thought, if not of deed—and have encouraged the wrong idea. In this connection it is interesting to note the relation of hoarding to pilfering. Misers and other "collectors" are prone to pilfering. I was consulted recently about a young woman who, in less excusable circumstances than most, took money belonging to others and hoarded it. When a girl she was unusually fond of money and frequently appropriated coins and hid them in strange places, mostly in books. Treatment is difficult. All cases require moral discipline, as well as physical bracing. Punishment of ordinary kinds will do harm in genuine cases when there is some degree of tumultuous impulse perhaps amounting to an epileptic equivalent. But patients who encourage wrong impulse may be deterred by suitable corrections. Pilfering from comrades is severely punished in the army and is comparatively rare.

SYSTEMATISED INSANE PURPOSE.—We need only glance at this form of mental disorder. it is not a recognised form of insanity, and the practitioner will seldom have to treat a private case. But it will be well if he recognises it as an insanity, and forms an opinion as to how to deal with it. For the patients involved will sooner or later become the subjects of legislation or of provision by our courts.

A systematised insane purpose is a deliberate intention to achieve some definite end by insane means. It differs from impulse in that it is a deliberate and abiding purpose, not a passing crave; and it is usually the expression of a conviction which may or may not be a delusion. A paranoiac, for example—and degenerates and paranoiacs are most subject to this form of madness—may be convinced that some one is his arch-enemy, and may consequently plot murder and deliberately set about to achieve it. Many paranoiacs conceive projects which are much more insane than the delusions of which they are the outcome. Vindictiveness, or at least the supposed redress of wrongs, is the motive in nearly all such cases. Very often there is a real and serious grievance. The insanity lies in the method adopted to achieve the desired result. And we must judge of such an insanity by taking into account the tradition and customs of the time. Sanity is the capacity of the individual to appreciate, and to regulate his conduct according to, the accepted standards of his day. An old-world Corsican, for example, whose family had suffered great injury or insult, who undertook a vendetta and slew the offender's grandson, would not, in his day, have been necessarily insane. But such an one who to-morrow revenged a real or supposed injury by stabbing his victim in the streets of London, must needs either be hanged as a murderer or detained as a dangerous lunatic. The motives of such insane revenge are usually either personal, or religious, or political. Insane purposes are often entertained by weak-minded persons who have a personal grievance. Others conceive that God or Allah, or some other deity, desires the death of certain people, and set about to accomplish it. But political crimes are those with which we are most familiar. The revolt of the Boxers in China and their intention to massacre Europeans would be an insanity in point, did they not represent just about the level of Chinese thinking and of Chinese morals. Nihilism is of this nature. And perhaps the best examples are to be found in anarchists, members of a civilised and law-abiding community, who set out to put wrong things right by insane and often violent means. From the point of view of mental science, political crimes are in no way justified by the fact that the patient entertains lofty sentiments about the rights of man any more than we would overlook the insanity of a patient who slept with his wife and family in the open fields because he entertained some enlightened views about tubercle. Much has been made of the love of notoriety as an important factor in political crimes. Its importance is probably exaggerated. The essential facts in such cases are that the intelligence is weak, so that the patient cannot think straight or see the various sides of his question; that he is possessed by disordered feelings of resentment which leave no room for justice and mercy towards innocent individuals; and especially that he has suffered an arrest of development which is obvious in his disability to believe in, and have patience with, constitutional methods of redress. In any case, the only possible treatment of such social insanities is by stern preventive measures. If anarchists and such-like criminals were quietly consigned to a criminal lunatic asylum their craving for notoriety would soon dwindle; and if this form of insanity were generally recognised, if it were understood that any one who advocated insane methods was *ipso facto* a lunatic, and a fit subject for asylum treatment, the mad crimes with which the world is so often horrified would materially diminish.

DEMENTIA.—All uncured insanities which are not terminated by early death result in some degree of dementia. After a few years, the longer an insanity lasts the more purposeless and silly the mind becomes. Primary dementia has been referred to, and, in some cases, a consecutive dementia, greatly resembling it in onset and course, follows soon upon an acute melancholia, stupor, or mania. Indeed, as was said, the acute insanity often seems to be a mere incident in the mental dissolution which is imminent. For that is what dementia is—a reduction of mental faculties; and it must be distinguished from states of imbecility in which the mental functions have never properly developed. A thorough dementia is a premature and rapid dotage. The various functions gradually dwindle, and the patient apparently does not think, is incapable of emotion, has no purposes, but is merely a creature of habits. Then his motor functions suffer; no thorough dement is capable of any work which requires skill: the sensibilities become blunt; the trophic functions are impaired, and the organic reflexes; and the patient slowly dies.

A great many dementias, however, are not thorough, but partial. The distinction is so obvious that one is inclined to think that every case of rapid and thorough dementia is an idiopathic dissolution, a premature senility, ushered in perhaps by an acute insanity. But the great majority of dements are only partially demented. The disease is limited, and it is not progressive. It is impossible to classify these secondary degenerations, for any one mental function may manifest the chief failure or a disproportionate survival, and in every case there is some general reduction, as well as an extreme degree of particular reduction. But these partial dements are interesting because they, more than most of the actually insane states, illustrate a reversion to primitive modes of consciousness. Every asylum, for example, can produce cases of insane constructiveness—patients who spend their days in harmless but quite puerile activity. Others are insanely predatory, or shamelessly acquisitive, and annex useful and useless things as indiscriminately as a magpie. Others spend hours daily in barbaric forms of self-adornment, or revel in vain exhibitions of themselves with ludicrous affectation. The sexual and the domestic instincts possess many partial dements, and these patients delight in promiscuous and often harmless flirtation, or in the furnishing of their rooms with ridiculous and lavish ornaments. In short, a partial dement may manifest any form of absurd and futile activity. For a general dementia is always notable in these cases—a decay of sane personality and a disability for serious and profitable occupation.

To the last, then, insanity is a systematic disorder. Subconsciousness is arranged and orderly, true to itself, though perhaps not suitable to a polite environment. In acute insanities, when sane personality is for the time being in abeyance, and in slow or partial dementia when it is for ever lost, the instinctive feelings and ideas and impulses of which subconsciousness consists, and which are a survival of ancestral or of personal experience, take its place and determine insane character. Yet is that character always intelligible and systematic—as orderly as the consciousness of a child, of a savage, or of a lower animal, and as certainly represented in nervous organisation.

VI. INDEX OF ETIOLOGICAL VARIETIES OF INSANITY

Dr. Urquhart[1] has purposely omitted an account of the forms which insanity often takes appropriate to the particular causes which he describes. Several of the etiological varieties are described in special articles. But it may be useful to suggest others. Although these are not all so constant as to be recognised as typical insanities, the practitioner may find it useful to know what forms mental disorders usually take, given certain etiological conditions.

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[1] Page 292.

(1) Idiopathic.—Imbecility (see "Mental Deficiency"); Degeneracy; Primary Dementia (see above); Paranoia (*q.v.*).

(2) Occasioned at critical periods of life.—Adolescence (*q.v.*); Pregnancy (*q.v.*); the Climacteric (*q.v.*); Senility (*q.v.*).

Insanity of Maturity.—Occurs when the patient incurs heavy responsibilities. Anxious melancholia, often proceeding to acute mania.

(3) *Persuaded Insanity.*—(*a*) Folie à Deux—A deluded patient persuades another to believe him; separation usually cures the second. (*b*) Folie à Plusieurs—Several persons may follow an insane lead. Babcock (*Amer. Journ. of Insan.* April 1895) quotes a good case where a household and several neighbours acquiesced in a systematised delusion of persecution. (*c*) Epidemic Insanity—Excitement, superstition, odd behaviour, frequently signs like saltatorial spasms.

(4) Toxæmic and Dyscrasic Disorders:—

(*a*) Stimulants, narcotics, and grain poisons. *Alcoholism* (*q.v.*); *Chloralism, Cocainism, Morphinism* (see "Morphinism"); *Ergotism.*—Gastro-intestinal and vascular disorders, varied mental symptoms often like general paralysis because of motor affections. *Pellagra.*—Stupidity, irritability, deep depression, delusions of fear. *Plumbism.*—Headache, insomnia, bad dreams, remitting delirium, or progressive delusional insanity.

(*b*) Specific Infections. *Typhoid*, etc.—Delirious and acute excited insanity in early stages, slow, depressed insanity often with motor signs in convalescent stage. *Influenza.*—The same, but motor signs often accentuated so as to resemble general paralysis. *Syphilis.*—Insane fear with delusions of syphilis when none exists, delusional melancholia in early stages, in later stages (tertiary and onwards) progressive dementia, or delusional suspicion and impulsiveness, or epileptic insanity, or tabetic insanity of anomalous forms, or resembling insanity of deprivation, or pseudo-general paralysis, or general paralysis. *Tuberculosis.*—In active phthisis, suspiciousness, moroseness, quarrels, fickle melancholia with mild delusions, or with hysteria, or with stupor, and, later, acute mania, or suspicious melancholia often with refusal of food and impulsiveness, with masking of physical signs, and, if not recovered, ending in fitful, asocial, querulous, semi-dementia; in slow tuberculosis, especially if abdominal, visceral melancholia. *Septicœmia.*—Acute, confused, delirious insanity, often recoverable, or ending in death by exhaustion, or going on to chronic mania with delusions and hallucinations (this includes some surgical cases and some puerperal cases).

(*c*) Constitutional Diseases. *Gout.*—In active gout, angry melancholia, or delirious mania (especially in over-use of sedatives), in "retrocessed gout," violent insanity perhaps epileptoid, in "suppressed gout," slow insanity often with hallucinations, in "undeveloped gout," neuralgic, neuritic, hysterical excitement, or moodiness. *Rheumatism.*—In acute fever, cf. "Typhoid," later a recoverable mania, melancholia rarer, or a slow amnesic, anergic insanity. *Diabetes.*—Melancholia with delusions of fear in persistent diabetes, or acute mania with temporary disappearance of glycosurea. *Urœmia.*—Fearful melancholia, great dread, exacerbations of violent excitement, impulsiveness.

(*d*) Affections of Nervous System. *Shock* (includes some puerperal and some surgical cases).—Confused, excited insanity with affection of memory, fancifulness, and perhaps hallucinations. *Traumatism.*—Moodiness, suspicion, outbursts of excitement, amnesia, motor signs, altered reaction to drugs. *Sunstroke.*—Neurasthenia, debility, motor or sensory signs, altered reaction to drugs, headache, melancholia and hypochondriasis, oftener exaltation and resemblance to general paralysis. *Neurasthenia.*—Various insanities. *Hysteria.*—Vain mania, often erotic. *Epilepsy* (*q.v.*). *Chorea* (*q.v.*). *Apoplexy.*—In later stages, excited insanity, amnesia, great proneness to laughing and crying, impulsiveness, violence, dementia. *Deprivations* (of hearing, sight, etc., and should include *aphasia* and some other lost movements).—Vexation, self-consciousness, dread, irritability, confusion, suspicion, excitement, delusion; melancholia rarer. *General Paralysis* (*q.v.*). *Tabes.*—General paralysis, or, not infrequently, pseudo-general paralysis, or anomalous insanity with chronic exaltation and exacerbations of violence.

(*e*) Disorders of Digestive System. *Para-digestion.*—Various insanities.

Dyspepsia.—Melancholia with hypochondriasis. *New Growths.*—Visceral melancholia, with delusions of suspicion.

(*f*) Disorders of Respiratory System. *Phthisis* (above). *Asthma.*—Recurrent mania or chronic mania. *Pneumonia.*—Cf. "Typhoid."

(*g*) Disorders of Heart, Vessels, and Blood. *Cardiac Disease.*—Melancholia with delusions of fear; in aortic disease, violence and variability commoner; dementia early. *Atheroma.*—Excited suspicions, melancholia, dementia. *Aneurysm.*—Visceral delusions. *Anæmia.*—Melancholia in men and in mature women; in young people, stupor or fickle, fanciful, excitable insanity with erotic and hysterical symptoms. *Myxœdema* (see "Thyroid"). *Cretinism* (*q.v.*).

(*h*) Affections of Reproductive and Genital Organs. *Ovarian and Uterine Disease.*—Recurrent insanity, delusions of sexual misfortune with melancholia, or delusions of exalted marriage with mania, modification of secondary sexual characteristics (beard, etc.); (delusions of a disagreeable kind often attach to some one person in the neighbourhood). *Functional Excess.*—Curable stupor, or progressive dementia. *Prostatic Disease* and *Stricture.*—Vexation, irritability, dread, excitement, sometimes going on to delusions of suspicion.

Insanity, General Treatment of.

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The General Treatment of Insanity.—Disease is known by the failure of function to satisfy the need of the organism. Treatment of disease is the endeavour to readjust the balance between function and need. There are two methods by which this balance may be restored. The impaired function may recover its efficiency, so that it again suffices to satisfy the need, or the need may be reduced until the impaired function is sufficient to satisfy it.

The restoration of function by artificial interference is seldom possible. Instant restoration, when it can be effected, is almost exclusively the privilege of the surgeon. He can instantly reopen the lumen of a hollow viscus whose obliteration prevented the passage of the visceral content. He can remove the tumour or evacuate the abscess, or cut the constriction which is interfering with function. He can tie the artery whose wound allows of the escape of that blood which it ought to contain. To the physician the direct restoration of function is less often possible, and when possible has rarely the dramatic suddenness and completeness which are achieved by the successful effort of the surgeon. The disturbing agent whose removal he attempts is rarely of the gross mechanical nature that so often has to be dealt with by the surgeon. Often it is some subtle poison to which he possesses no antidote; often it is some diffused and microscopic degradation of tissue whose restoration, even if the degradation can be recognised, is beyond the resources of his art. Only when he possesses an antidote, only when he can neutralise the poison by the administration of some appropriate drug, as by mercury or iodide of potassium in syphilis, or by an appropriate antitoxin in a specific fever, can he pursue the direct method of restoration of function in the former class. In the latter he is altogether powerless to pursue this method. All that he can do is to reduce the need to the level of the impaired function. When the aeration of the blood is impaired by disease of the respiratory organs or of the heart, he reduces the need for aeration by forbidding exercise. When the digesting power of the stomach is impaired, he reduces the need for digestion by reducing the quantity of food, or by administering food easy of digestion or already partly digested, or by administering digestive agents with the food. When the kidney fails in its power of eliminating nitrogenised waste products, he diminishes the need for such elimination by diminishing the ingestion of nitrogenised food. When writing is rendered impossible by writer's cramp, he diminishes the need for writing by ordering the use of a typewriter. When the function of a joint is impaired by inflammation, he keeps the joint at rest. In any case he reduces the need for the function as far as possible down to the capacity of the function impaired, and thus restores the balance between function and need.

It is evident that this second mode of restoring the balance is a poor substitute for the first. The restoration of the impaired function to its normal standard is a true *restitutio ad integrum*, and brings the organism back to its full measure of capacity. The reduction of the demand of the organism down to the level that the impaired function can reach is at best a makeshift, and an acknowledgment of our impotence to do better; but this second mode of restoring the balance is not without its influence on the first. The reduction of demand upon an impaired function influences the restoration of the function in both a favourable and an unfavourable direction. On the one hand, the reduction of demand upon the function, especially if the reduction can be pushed to actual cessation, sets, in the one case a part, and in the other the whole, of the anabolic process free to work at the restoration of the damaged structure, which otherwise would be engaged in the double process of restoring structure, and at the same time actuating function, a combination that is rarely within its capacity. When the hull of a ship is damaged, she must be brought into dock for repair; when her engines or her boilers are outworn, they cannot be made good while she is on active service; and similarly the tissues of the human body cannot undergo repair so long as full functional activity is demanded of them. Only during intermittence of function can redintegration progress; and hence those tissues, in which waste is most rapid and function most exhausting, need the frequent functional cessation of sleep to keep them in repair. The reduction of demand upon an impaired function is therefore a most important condition for the restoration of the function, and is in very many cases the chief, in many cases the sole, means at our command of bringing about the restoration of the function. On the other hand, no facts in physiology are better established than that function tends to increase with the demand that is made upon it, and that prolonged diminution of demand is followed by diminution of function. From the arm of the blacksmith to the verbal memory of the actor, we see how demand increases function; from the leg of the omnibus-driver to the intelligence of the soldier, we see how lack of demand leads to cessation of function. Hence we are taught that while on the one hand, as soon as a function is impaired by disease, the demand upon it should be immediately reduced to the lowest possible level, yet on the other hand, as soon as the process of repair is well on the way, the demand should be cautiously increased, so as to lead forward and assist in the re-establishment of the function.

Applying these principles to the special case of insanity, we find that the treatment of that, as of all other maladies, is twofold. The main end in view in every case must be the restoration of function to its full capacity, and as a means to this end the demand upon the function must be reduced, at first to or below the level of the capacity of the impaired function. As and when improvement of function takes place, the demand upon it must be cautiously raised, and kept always just a little in advance of its capacity, so as to lure it by degrees to wider flights and stronger efforts. Even if no improvement appears to indicate the period at which increasing demands may be made, still the increase of function must, after a time, be called for, and persistently called for, such demand being commonly the only means at our disposal of eliciting an improvement in function.

Seeing that of these two modes or aims of treatment of insanity the second is always possible, is always imperative, and the mode of effecting it usually clear and simple, it may be dealt with first; while as the other can be pursued only tentatively, by groping in the dark, and is often, in the present state of our knowledge, not possible at all, its consideration may be deferred.

THE REDUCTION OF FUNCTION.—The function which in insanity is disordered and deteriorated is that of the supreme regions of the nervous system, by which conduct is actuated, by which the metabolism of all parts of the body, both absolutely and relatively to each other, is regulated, and which is accompanied by states and changes of consciousness. Of these functions the first alone is open to our direct interference. The others we can modify indirectly only. The need for that adjustment, by intelligent acts, of the organism to its surroundings, which is termed conduct, we can diminish almost *ad libitum* by furnishing a set of surroundings so completely preadjusted by artificial means to the needs of the organism, that voluntary adjustment can be dispensed with. All the more intelligent phases of conduct, and, if necessary, all phases of conduct but the most rudimentary, can be supplied by artificial substitutes, and the highest nerve regions, thus relieved of their functions, can be left at rest to deal with the disturbing agent that is acting on them, to recuperate their deteriorated structure, untrammelled by the need of simultaneously carrying on their function.

In all cases of insanity, or of defect or disorder of mind or of conduct, which, though of the same nature as insanity, would not be called by so serious a title—in all cases of such disorder, whether incipient or established, whether mild or aggravated, the first need is the need of rest, of reduction or abolition of the demand upon the disordered function. The recognition of this need has sometimes led to the pursuit of an exaggerated course, and has been interpreted to mean that every such patient, whatever the form or degree of his malady, must be deprived as completely as possible of all opportunity of conduct, by being put to bed. This is as intelligent a mode of treatment as to put to bed every patient with a broken bone, regardless of whether the bone broken is that of a leg or an arm or a finger. "He physic's use doth quite mistake, who physic takes for physic's sake." If those parts of the nervous system, which regulate the most complicated relations of the organism with its surroundings, are unequal to the demand upon them, and cannot regulate their relations efficiently, that is a reason for reducing or removing the demand upon them; but if inferior portions are still capable of carrying on their functions with efficiency, they may still be permitted to do so, even though their superiors must be relieved. If the apparatus by which a man carries on his business is disordered, it is right and necessary that he should be relieved from the opportunity of carrying on his business; but it is no more necessary that he should therefore be relieved from the opportunity of walking about and amusing himself than that he should be relieved of the opportunity of voluntarily emptying his bladder. If every insane person is to receive what is humorously termed "hospital treatment," that is to say, is to be kept in bed, the treatment is not consistent unless he is also relieved by a catheter and fed with a stomach-pump.

The difficulty of suspending or reducing the call upon function in insanity lies in the differences between insanity and other maladies. If a man suffers from any bodily malady, he seeks, his conduct not being disordered, the advice of some competent authority, who gives the advice and leaves to the patient the responsibility of acting upon it. But in insanity it is conduct itself which is disordered, together with mind, and hence in the first place the patient very often does not know that he is suffering from any malady; and in the second, if he do know, he is often unable, from the disorder to which his conduct is subject, to take the course with regard to it which even he himself may see to be expedient. For this reason it is often necessary in insanity to act without the consent, and even in opposition to the wishes, of the patient, and to deprive him forcibly of those opportunities, which he will not willingly relinquish, of exercising his disordered faculties.

It is often necessary, but it is far from being, as is sometimes supposed, always necessary. In the case of minors, they can be taken away from the school or the college or the course of studies in which the excessive demand upon the immature brain has caused the break-down. In the case of the adult, he can often be persuaded into, nay, will often himself suggest, a temporary retirement from active employment, and a season of fallow, when he feels that his capacity is no longer equal to the demand upon it; and in mild degrees of the malady, and in these only, such measures are sufficient; but not only are such measures impracticable in the case of those who will not submit to them; they are also often inefficient even in the case of those who will. A man who knows himself unfit for business, who feels his incapacity to follow his profession, may voluntarily intern himself in an asylum, and go from bad to worse until it becomes necessary that he shall be certified and compulsorily detained; and from the day that this is done he begins to improve and makes steady progress to recovery. It seems as if the mere responsibility for determining his own course of conduct were too heavy for him to bear; that he was weighed down by the uncertainty as to whether he was doing right; and that only when this responsibility was entirely removed by his compulsory seclusion from the ranks of those *sui juris*, only when he could feel that he was relieved of all responsibility for the regulation of his own conduct, was obtained that complete quiescence of the highest nerve regions which rendered recuperation possible.

To take over by force the regulation of a man's conduct, is a proceeding which lies entirely outside the province of the physician. Such a proceeding is *ipso facto* a menace to every member of the community. It is one which is not to be countenanced except upon the strongest grounds of expediency, and it is one, moreover, which the community will not and ought not to permit without the sanction of its own accredited representative, given under specified conditions laid down by itself. These conditions vary very widely in different communities, and even in the several divisions of these islands they exhibit a surprising diversity. Broadly, it may be stated that the formalities whose observance is required in depriving a "person of unsound mind" of the regulation of his own affairs are divisible into two classes, those which refer to the deprivation of his management of his means of livelihood, and those which refer to the deprivation of his management of the rest of his relations with his surroundings. His means of livelihood are called in English law his estate, while the maintenance of due relations between himself and his surroundings in other respects—between himself and physical dangers, between himself and his family, between himself and the community in all respects other than his means of livelihood—is regarded as peculiarly the management of himself. With the formalities which are necessary for the deprivation from a person of the management of his estate, the physician is not directly concerned, and they need not be considered here, further than by mention of the fact that, under the English law, a person may be deprived of the power of managing his estate, while he may be at the same time left in undisturbed possession of the management of all his other relations with surrounding circumstances.

The formalities necessary for depriving a person of the management of these residual relations differ, as has been said, in the three divisions of the United Kingdom, and they differ in each division, moreover, according to the degree to which the deprivation is carried. For degrees of deprivation are made, and a person may be deprived of the management of more or fewer of his relations with his surroundings, or, as we may say more briefly, of his affairs, according as it appears expedient. It is very much to be regretted that in the laws of England only the grosser degrees of incapacity can be dealt with by deprivation of power. Every year, nay, every month, and almost every week, occur cases in which a goodly estate is dissipated, an honoured name is besmirched, an innocent family is brought to scandal and to poverty, by the prodigality, the vice, the folly of some unworthy scion who is incapable of prudence and rectitude, but whose incapability is not of such a nature as to enable his relatives to put the provisions of the Lunacy Acts into force for their own protection. There is not an alienist physician, there is not a family solicitor in large practice, nay, there is not an intelligent reader of the law and police news in the daily papers, who is not aware of cases of this kind, in which, if not scandal, at least impoverishment could have been averted, had the excellent provisions of the French *conseil de famille* been in force in this country. However, our concern here is with the law not as it ought to, but as it does exist, and with this preface we will proceed to state it as it stands.

The legal provisions under which a person can be deprived of the regulation of his own affairs differ, first, according as his defect in regulating power is congenital or not; and, second, according to the amount of his property.

ENGLAND AND WALES.—An *idiot or imbecile* from birth or from an early age may be placed under control, and detained in an institution registered under the Idiots Act, upon the certificate of one medical practitioner, accompanied by a statement made by the parent or guardian of the patient or by the person who undertakes towards him the duty of a parent or guardian (Idiots Act, 1886, sect. 4). As these documents are of a very simple character, need no comment, and are obtainable at any registered institution for idiots, there is no need to introduce them here.

A lunatic *wandering at large* must be apprehended by the police and brought before a justice, who will send the patient to the county or borough asylum of the district. Either before or after he is so sent his friends may obtain control of him by making application, in the one case to the magistrate, in the other to the visitors of the asylum and to the Commissioners in Lunacy.

A lunatic *who is not under proper care and control, or who is cruelly treated or neglected* by those who have charge of him, must be reported by the police on oath to a justice, who will proceed as directed in the Act.

A lunatic *who is a pauper* must be reported to the relieving officer of the district by the union medical officer of his district within three days of the medical officer obtaining knowledge that the pauper patient is or is deemed to be a lunatic.

For a lunatic *who is not a pauper* the procedure is various.

The patient may, if he is willing to do so, and if he is competent to form a judgment as to the expediency of doing so, enter a licensed house as a voluntary boarder, by previously obtaining the consent of one of the licensors—of a commissioner in the case of a metropolitan licensed house, or a visiting justice in the case of a provincial licensed house; or he may enter as a voluntary boarder a registered hospital without any previous consent other than that of the manager of the institution. In any case he must not be so insane as to be certifiable.

A bill is now before Parliament authorising the temporary admission into private care of a lunatic whose insanity is not confirmed, under a single medical certificate and without any further authority. This provision is adopted from the Scotch law, which see.

If the patient is certifiable, and if his malady is "confirmed" (see *infra*), there are three modes by which his detention under care and treatment may be legalised: the judicial reception order, the urgency order, and the inquisition.

The judicial reception order is obtained from a justice specially appointed under the Act, or from a stipendiary magistrate, or from a County Court judge, by means of a petition from a relative of the patient, accompanied by a statement of particulars and supported by two medical certificates. There is no need to give here the form of the medical certificate, which is sufficiently well known, nor is any accomplishment needed for its correct execution beyond ordinary care and ordinary intelligence. It is nevertheless remarkable how few certificates are correctly executed. The obvious precaution of referring to all the marginal notes and complying with the instructions that they contain is usually neglected, as is the other obvious precaution of reading it carefully through after it is made. The consequence of this neglect is that certificates, which are discreditable to their signators, are very frequently sent to the commissioners, by whom they are returned for the correction of palpable blunders which never ought to have been made. Apart from the form of the certificate, which can be made in due order by the exercise of the most ordinary care, the substance demands attention. The object of the certificate is to satisfy the mind of the judicial authority to whom the petition is presented, that the patient is of unsound mind, and a proper person to be detained under care and treatment. It should, therefore, contain such evidence of these facts as shall carry conviction to the mind of a person who has never seen the patient and knows nothing about him. Too often the medical practitioner looks upon himself as the only person who has to be satisfied of these conditions, and embodies in his certificate little more than an assurance that he is satisfied. This is a very erroneous view. What is required by the justice is not the conclusion at which the medical practitioner has arrived, but the grounds of this conclusion. The terms of the certificate itself are quite sufficient to indicate what is required. They require the practitioner to state that he has arrived at the conclusion that the patient is of unsound mind, etc., and that he is a proper person to be taken charge of and detained under care and treatment; and further, that "I have formed this conclusion on the following grounds, viz.: facts indicating insanity observed by myself at the time of examination," and "facts communicated to me by others." It would be difficult to put into more explicit terms the requirement of the law, and yet this requirement is frequently ignored. Very commonly, under the head of "facts indicating insanity," is inserted a diagnosis, and still more commonly is inserted an opinion which the certifier mistakes for an observed fact. The only "facts," rightly so called, which can properly be stated in a certificate are the sayings, doings, and in some cases the appearance of the patient. Seeing that the certificate states that the patient is of unsound mind, the certifier frequently falls into the error of enumerating states of mind as "facts indicating insanity." He will say that the patient is depressed, that he suffers from delirium, that he thinks this, and feels that, and believes the other. But these are not "facts observed at the time of examination," or at any other time. They are opinions. They are inferences drawn from the sayings and doings of the patient; and it is these sayings and doings, and not the inferences from them, that are in fact observed, and that alone may rightly be included in the certificate. If the act or statement of the patient is manifestly insane; if, for instance, he hands his interlocutor a scrap of newspaper on which is written a cheque for a million pounds; or if he knocks off his visitor's hat and jumps upon it, then no more than the bare recital of the fact is needed. But if the act or statement is only conditionally insane; if, that is to say, it cannot be known to be insane without a knowledge of the circumstances of the patient, then so much of these circumstances as suffice to establish the insanity of the act or statement must be embodied in the certificate. If, for instance, the patient states that he is ruined, or that his leg is broken, or that his wife has deserted him,—statements which are only conditionally insane,—then the practitioner must satisfy himself, first, that the statement is untrue; second, that the patient cannot be convinced of its untruth; and must state these qualifying facts, as well as the primary fact of the statement itself, in the certificate.

The disadvantage of the procedure by judicial reception order is that, requiring as it does the concurrence of several—at least four—persons, it necessarily occupies time, and sometimes the necessity of taking out of a person's own hands the control of his affairs is so instant and importunate that this time cannot be given. For these cases is provided the procedure by urgency order, under which it is lawful to detain a patient for a limited time upon the order of the relative, accompanied by a medical certificate, not only of his insanity and of the evidence of it, but likewise of the reasons which render it expedient that he should be forthwith placed under care without waiting for a judicial reception order. The reason for urgency may be either the welfare of the patient or the public safety. When a patient is detained under an urgency order he must be released at the end of seven days unless in the meantime a petition for a judicial reception order has been presented to a judicial authority; but if within seven days such a petition has been presented, then he may be detained under the urgency order until that petition has been disposed of. There is at the present time a measure before Parliament which, if it becomes law, will reduce the period of independent validity of the urgency order from seven days to four.

The inquisition is a measure to which recourse is had only when the property of the lunatic is considerable, or when he demands an inquiry into his sanity with a view to his release. It is of the nature of a trial at law, and the function of the physician with regard to it is merely to give evidence when called upon. An inquisition results, if the petitioner is wholly successful, in the appointment of a committee of the person and of a committee of the estate of the lunatic, who are responsible to the court for the proper performance of their duties. Each committee may consist of one or more persons, and the same person or persons may act in both capacities. It is competent to the court to find that the lunatic is unfit to manage his affairs, but fit to manage himself, and in that event no committee of his person is appointed, and his personal liberty is not restricted.

Scotland.—Idiots and imbeciles under 18 years of age may be received into training schools in Scotland without certification or other legal formality. In practice, however, pauper children are invariably sent thither under the same formalities as are used in the case of adults sent to other institutions for lunatics, with the addition that the consent of a commissioner must be first obtained. The reason for this procedure is that when so sent only is the school entitled for each child to a 4s. grant. When above the age of 18, and when sent to institutions other than training schools, idiots and imbeciles are in Scotland classed as lunatics, and are subject to the same formalities.

Insane persons whose malady is not confirmed may be placed in private care for a period not exceeding six months, under the special certificate of one medical practitioner, and without other formality.

The ordinary procedure in pauper and non-pauper lunatics is the same, and requires a petition to the sheriff accompanied by a statement of particulars and the certificate of two medical practitioners. In the case of a pauper the petitioner is the inspector of poor.

If the case is urgent, a certificate of emergency by a single medical practitioner suffices to authorise the detention of the patient for three days. This certificate must be accompanied by a request, from the person in the position of petitioner, to the superintendent of the asylum, to receive the patient.

In addition to these provisions, the Scottish law provides a process termed *interdiction*, which has no counterpart in English, though it resembles certain proceedings in French law with which it has a common origin in the *jus civile*. Interdiction is a legal restraint or incompetence fixed upon persons who are prodigal, or are too easily influenced by others, and disables them from alienating their property without the consent of guardians, who are called interdictors. Interdiction may be voluntarily entered into by the interdictee, or may be compulsorily enforced upon him by the Supreme Court, and in the latter case the decree of the court may be granted either upon the initiative of the relatives of the *prodigus*, or upon the initiative of the court itself, founded upon its own observation of the conduct of a party to a suit before it.

The proceedings of cognition and curatory are analogous to the inquisition in English law, from which, however, they have wide differences. If successful, they result in the appointment of a committee, or, as he is called, a curator, to administer the estate of the lunatic; but there is no functionary corresponding to the committee of the person of the lunatic.

Ireland.—The formalities differ according as the patient is to be taken to a district asylum on the one hand, or to a licensed or unlicensed private house on the other.

Pauper patients who are not dangerous are admitted *into a district asylum* upon four documents:—1. Declaration before a magistrate stating that the patient is insane and destitute, and giving names, etc., of two relatives. 2. Certificate of a magistrate and a clergyman, or poor law guardian, that they have personally inquired into the case. 3. One medical certificate. 4. An engagement by the applicant to remove the patient when called on.

Paying patients who are not dangerous require five documents to authorise their admission into a district asylum:—1. Declaration before a magistrate that the patient has not sufficient means to pay for support in a private asylum, and has no friend who can do so, and stating how long the patient has been resident in the country. 2. Certificate of a magistrate and a clergyman, that the case has been investigated. 3. One medical certificate signed by two medical practitioners. 4. An engagement to remove, together with an engagement to pay a specified sum. 5. The sanction of an inspector of lunatics.

The proceedings above described are so cumbrous that in practice the great majority of patients in the district asylums of Ireland are admitted as *dangerous lunatics.* Any person may be apprehended "under circumstances denoting derangement of mind and an intention of committing an indictable offence," and removed by warrant of the Lord Lieutenant from gaol to an asylum.

Into *licensed houses, charitable institutions, and single care,* patients are admitted upon the following documents:—1. An order by a relative or connection of the lunatic. 2. A medical certificate of two medical practitioners.

In cases of urgency the signature of a single medical practitioner is sufficient, provided that a second be added within fourteen days of the first.

Finally, the proceedings under *inquisition,* which are practically the same as in England.

The Advisability of Asylum Treatment.—From the foregoing account of the methods of procedure in force for the legal deprivation from persons of unsound mind of the management of their affairs, it will appear that several degrees and modes of deprivation are recognised and sanctioned by the law, and the first question that will present itself to the practitioner, when he has determined that his patient is of unsound mind, will be, Which of these degrees and modes is proper to be adopted in this case? Ought the patients to go to an institution, or to be placed in private care? and in either case ought he to be persuaded to go, and ought his remaining there to depend upon his own free will as it is influenced by moral suasion, or ought he to be deprived of his liberty by due process of law?

The decision must depend upon several very diverse considerations, of which the chief are—The nature of the malady, the age of the patient, and his means.

1. *The Nature of the Malady.*—A patient who is definitely suicidal or acutely maniacal ought to be at once sent under certificates to an institution. To this rule there is no exception. It is only in an institution that the structure and appliances of the building are specially arranged for the accommodation of such patients. It is only in institutions that the windows, the appliances for lighting and heating, the staircases, the water-closets, the bedsteads, and a score of other arrangements, are specially adapted to obviate the suicidal or dangerous or destructive proclivities of the patient; and although it is, of course, within the competence of persons of sufficient wealth to adapt the structure of their houses and the contents of their houses in a similar way, it is not competent to them to effect these alterations with the necessary speed to secure the immediate safety of their relative. Moreover, such patients need, if not the continuous services of a medical attendant, at any rate the continuous presence of a medical attendant within call; and this is scarcely to be obtained at a moment's notice in a private house. Even in the case of a man of unbounded wealth, who could secure the services of an unlimited number of experienced attendants of all grades, the element of time would always

necessitate the immediate removal of the patient to an institution as a first step. The rule admits, therefore, of no exception. But the question immediately arises, How are such patients to be recognised? With respect to the maniacal patient there is no difficulty. The degree of his excitement, the outrageousness of his acts, at once place the matter beyond doubt. But with the suicidal patient the difficulty is often great. A definite attempt at suicide, of course, settles the matter. After that there can be no doubt. But it by no means follows that a melancholic patient who has made no attempt, and no verbal reference to suicide, has yet not formed a determination to take his life on the first opportunity; and it often tests to the uttermost the diagnostic power of the most experienced expert to determine in any given case whether the tendency exists or no. As a rule, the non-expert greatly under-estimates the likelihood of a suicidal tendency. The expert, on the other hand, tends rather to over-estimate it. But then the latter has probably seen such disastrous results from the under-estimation that he deliberately chooses to err upon the safe side. Whenever melancholia is decided, is pronounced, and whenever mania is acute, and is unattended with exaltation, a suicidal tendency should be strongly suspected, nay, should be "conclusively presumed," and measures should be taken accordingly. It is not said that where these features are not present the suicidal tendency will be absent, but that where they are present it may be presumed.

Where neither suicidal tendency nor acute mania is present, the question of treatment in an institution will depend upon the amount of control that it is necessary to exercise. Where the amount of control needed is not great; where the patient does not need very vigilant supervision; where his disorder of conduct is not of such a nature as to imperil his own safety or that of others; is not displayed in sexual matters; is not such as to render him very conspicuous; is displayed only at intervals, and on occasions that can be foreseen and provided against; then he may be properly treated in private care. But when the conditions are the reverse of these he ought to be placed in an institution. There the amount of control that can be exercised, and that ordinarily is exercised, is much greater than in private care, and therefore that is the proper destination of patients for whom much control is needed. For this reason patients with delusions of persecution are unsuitable for private care. Every such patient is a potential homicide, and requires an amount of control that need not be very strict, but that must be continuous; and continuity of control can be secured much more certainly in an institution than in private care. Generally, the graver the disorder of conduct, the more pronounced the malady, the more suitable is the case for treatment in an institution; and *vice versa*.

The *age* of the patient is a factor of some importance in coming to a decision. We are very reluctant to send a young girl or a lad to an institution for lunatics if such a course can be avoided. So long as the savour of the "madhouse" clings to these institutions in the mind of the public, so long as any remnant of that semi-superstitious horror remains with which the ignorant public is apt to regard them, so long we must avoid, if it be possible to avoid, sending to them young people who have all their careers before them, and whose future lives may be more seriously affected by the reputation of having been in a lunatic asylum than by the reputation of having been unsound in mind.

Lastly, the *means* of the patient is a factor of considerable importance. Private care is as a rule a more expensive mode of control than care in an institution, and when the amount of control that is needed is great the disproportion is correspondingly great. A patient who needs continuous supervision both by day and by night, will in private care need the services of at least two attendants, and if the supervision is prolonged, of three; and attendants whose services are temporarily engaged, as in private care, demand much higher wages than those whose engagements are permanent. In an institution the same patient would not need the exclusive services of these attendants, and each attendant would be less costly; so that if expense is a consideration the institution is to be chosen.

As these are the main factors in determining whether the patient shall be sent to an institution or treated in private care, so are they the main factors in determining whether resort shall be had to legal process for restricting the power of the patient over his own conduct.

If the case be one of mere dementia no such resort is needed, unless for reasons of expense or convenience it is determined to send the patient to an institution, and the sending must be legalised. Mere dements are already deprived by their malady of the control of their conduct; and no intervention is necessary to do what the disease has already done. All that they require is the same supervision and nursing that is required by children of equivalent intelligence. In the case of minors whose conduct needs more active control, certification can usually be avoided, since they are not yet fully *sui juris*, and the authority of the parent to control their conduct does not, except in severe cases, need to be reinforced by the special provision of the lunacy law. If the parent chooses to say to his child, "I have made arrangements for you to leave home and to be placed in the care of So and so, to whose authority you must submit," the child has no alternative but to obey. When an adult is but slightly affected by unsoundness of mind, and is himself aware of his unsoundness, and desirous of placing himself under treatment, then of course he may make whatever arrangements he pleases for relinquishing the control of portions of his affairs. He may give power of attorney to whomsoever he pleases to deal with his estate, and he may take what advice seems to him judicious and desirable as to the disposal of other departments of his conduct. He may place himself in private care or in an institution as seems best.

Advice is often given, especially by neurologists who have no special knowledge of insanity, but are frequently consulted with regard to it, that the patient should travel, either by himself, or with a friend, or under the care of an attendant, medical or other. Such advice is rarely judicious. The object of suggesting a change of surroundings is to liberate the patient from the necessity of regulating his conduct in certain departments; to reduce the call upon function. But in travelling, while if he relinquishes the care of his estate, the call upon function is diminished in this direction, it is manifestly increased in others. The constant change of surroundings brings of necessity the constant need of changes of conduct in adaptation to the changes of surroundings, and the relief that is obtained in one direction is nullified by the increased demand that is made in others; so that we find in practice that in the early stages of mental disorder travelling is not beneficial. The cases in which it is most commonly advised are cases of slight melancholia, and the advice is given from the analogy of the relief from care that is gained by jaded men of business from a holiday with change of scene. But the two cases are not analogous. The jaded man of business has lost the elasticity of his spirits from a too complete engrossment in the cares of business,—from the circumstances in which he acts. His depression is due to, and proportionate to the stress of circumstances upon him, and when relieved from the stress of circumstances he recovers. But with the melancholic it is otherwise. However much the stress of circumstances may have contributed to the establishment of his malady, it is, when established, independent of his circumstances. The definition of melancholia is that it is a state of misery that is not justified by the circumstances of the patient; and therefore the mere relief from these circumstances is not sufficient to remove it. What the melancholic, with his conviction of his own incompetence, needs, is circumstances that make no demand upon his competence; and these circumstances are not to be found in travelling. Let him leave home by all means; but let him go to some one spot and there remain, in surroundings in which demands upon his faculty of regulating his own conduct are minimised. The greatest mistake of all is made when the melancholic is recommended a sea-voyage. Here he is placed in circumstances of dismal monotony, with the temptation to suicide constantly pressed upon his attention. What wonder if he often succumbs?

Regulation of Control.—Having complied with the legal formalities, and released the patient from the obligation of regulating for himself the more elaborate portions at any rate of his conduct, the next question in treatment that presents itself is, to what extent this release is to be given or this control to be exercised. This must depend upon the depth to which conduct is disordered. If only a superficial flake is peeled off from the upper strata of his capabilities,—if, for instance, as sometimes happens, his conduct is disordered in certain social relations only,—if his conduct say towards the members of his family, or towards members of the other sex, or towards religious ceremonies, or towards children, or with respect to legal proceedings, is the only region in which his conduct is disordered—then there is no need to exercise control over him except with regard to the particular relation disordered. There is no reason why persons whose disorder of conduct is limited to such particular divisions as those above specified should be deprived, for instance, of the management of their "estate," nor of other aspects of their affairs. The limitation of disorder of conduct and the limitation of control which logically follows upon it, are explicitly recognised both in English and Scotch law by provisions which allow the patient to retain the management of his other affairs, while he is deprived wholly or partly of the management of his estate; and there is no reason, save the difficulty of formulating in words the department of conduct disordered, and of precisely limiting the control to that department, why the principle should not be generally applied in statutory enactments. But if not applied in law, it can and ought to be applied in practice, and the principle upon which control should be exercised over lunatics should be, in all save acute and recent cases, that the control should extend over that region of conduct only which is disordered. This principle is already followed to some extent in practice, but it is followed partially, haltingly, and with no true perception of its full application. All patients who are detained as lunatics are not controlled in the same degree. Some are allowed their liberty on parole, others are not; some are allowed the restricted use of money, others are not; some are allowed to regulate their own costume in accordance with their own taste, others are not; most are allowed to take their own food in such quantities as they desire, others are forcibly fed; and other distinctions are in practice. But in most institutions and in every large institution there is a certain fixed maximum of freedom which no patient is allowed to exceed, and this maximum is far too low, and is fixed without regard to the considerable class of patients whose activities need restricting in one direction only, and in different directions for each.

To this rule, that conduct should not be controlled further than it is disordered, an exception has been made. In all acute and recent cases the control must be very complete; and this for several reasons. In the first place, until the patient has been under observation for some time it is not known with certainty how far conduct is in fact disordered. In the second place, it is in acute and recent cases that a principle already alluded to applies, viz. that the demand upon function shall be withdrawn very completely so as to leave the metabolic process at full liberty to restore the damaged structure. The validity of this exception is fully borne out in practice. It is found that in acute and recent cases the mere removal from home and compulsory detention among strangers has of itself, without further measures, a distinct ameliorative effect; and it is a well-established fact, known to all who have care of the insane, that during the early days of their malady the visits of relatives and friends with whom they are in habits of social intercourse has a detrimental effect. The beneficial influence of detention under control is often ascribed to the mere change of surroundings of every kind which it usually involves, and it is probable that a part of this beneficial influence is correctly so ascribed; but it is probable also that in the presence of near relatives and customary friends the patient feels that the control over him is relaxed, and that he may venture upon a degree of self-assertion which in their absence he would not aspire to. Be the explanation what it may, the fact is indisputable that in recent and acute cases of insanity the mere removal from home and the detention under care have of themselves a beneficial effect, an effect which is at once neutralised for the time being by the visit of near relatives. Fortunately the ill effect of those visits does not usually endure. Sometimes the melancholic who has poured out his woes to his wife and has wept copiously over his imaginary troubles, will cheer up on the instant of her departure and join in amusements as heartily as he was doing when her visit was announced. More often it takes him a few hours to get over it, but I cannot say that I have observed any permanent ill effect.

It is in cases in which the disorder or defect of conduct is deepest, in which the most fundamental and primary departments of conduct are affected, that the control must be most complete. Where the primary instinct of self-preservation is absent, or is reversed; in the deeper forms of dementia, in which almost all forms of conduct are lost, and the patient is reduced to the infantile condition; when he is incapable of preserving himself from simple mechanical dangers; when he cannot be trusted alone in a room for fear he should fall out of his chair into the fire, or stuff into his mouth any uneatable substance—coal, rag, or paper—within his reach; when, like an infant, he is inattentive to the state of distension of his rectum and bladder, and passes his excretion as he sits; or when, instead of using his activity to preserve his life, he endeavours with all his resources to put an end to it; in either case the control over him must be of the most minute and vigilant character. But the two cases are manifestly different. The one may be left to the nurse and the general physician. The other needs the utmost exertions of the most skilled attendants on the insane. The only chance of preventing a suicidal patient from carrying out his intention is incessant and minute vigilance. Even incessant vigilance is not always successful, but it is the only means at our disposal, and it must be employed.

When the case is no longer recent; when time has been given for any acute process of disorder in the brain to subside, and the stage has arrived for attempting to restore the deficient function, and to re-educate the patient into normal modes of conduct, then control must be gradually lessened, and the patient must be encouraged and stimulated to exercise those departments of conduct of which he has been hitherto deprived. So long as the bones of a broken arm are ununited, so long must the arm be kept at rest in a splint, and its function suspended. So long as the valves of the heart are the subject of acute inflammation, so long must the function of the heart be minimised by keeping the patient in absolute rest. But as soon as the bones are united we encourage the patient to overcome the stiffness of his fingers by using them; as soon as the cardiac inflammation has subsided we encourage the patient by graduated exercise to strengthen the muscle of his heart; and as soon as the acuteness of the outbreak of insanity passes away, we should encourage the patient to resume, tentatively and intermittently at first, but with gradually increasing frequency and completeness, the control of his own relations with his surroundings. It is at this time that the skill of the alienist is tested to its utmost capacity.

Other General and Special Treatment.—Leaving now the question of the regulation of conduct by external coercion, we enter on the consideration of the second branch of treatment, and ask what means we have of acting, by ingesta and in other ways, upon the disordered cerebral processes so as to bring about their restoration to the normal. We find that our knowledge of such means is much on a par with the knowledge that Voltaire attributed to metaphysicians,—"fort peu de chose." Something, however, we have gained.

Under normal circumstances the great restorer of the function of the highest regions of the brain is sleep. Towards the end of every day sets in an exhaustion of these regions which, if pushed to extreme, exhibits itself in disorder. No one is as active, as alert, as mentally competent, after a sleepless night as after a night of sound sleep; and the persistent deprivation of sleep of itself leads to great disorder. To this we add the knowledge that in all cases of acute and recent insanity sleep has recently been defective, and thus we gain a clear and indisputable indication for treatment in all such cases. Measures must be taken for the induction of sleep. And the propriety of the indication for treatment is shown by the result. Until sleep is induced improvement does not take place. When sleep is induced, improvement does, as a rule, occur. How, then, is sleep to be induced? The obvious reply is by the administration of an hypnotic drug, and for many years the routine administration of opium in recent cases of insanity was followed, and the natural consequences were such that a reaction, as unreasonable as the practice, set in against the administration of all hypnotics whatever, and a most pernicious maxim gained currency that "chemical restraint is as bad as mechanical restraint." It is difficult to say which part of this question-begging statement is the most to be condemned, whether the false analogy between sleep and restraint, or the implication that mechanical restraint is, *ipso facto*, and in all cases, bad. If a patient is sleepless; and if I have a drug the administration of which will produce sleep; and if after the sleep so produced the patient is better, is more normal both in mind and conduct; am I to be precluded from the administration because of the stupid and unjustifiable statement that I am restraining my patient by chemical means? Such slavery to a form of words would be unworthy of the most narrow-minded pedant that ever exercised his degraded ingenuity in Westminster Hall. But is it a fact that sleep so produced is ever beneficial? I affirm with the utmost certainty that it is;

and I go farther, and say that by the administration of drugs we have the power in certain cases of converting a furious maniac into a comparatively sane man; of reducing his maniacal fury down to mere restlessness, and even to tranquillity; and of freeing his mind, temporarily at any rate, from the delusions to which it was subject. This may be "chemical restraint," if we please to call it so, and if it be, let us be heartily thankful that the resources of modern pharmacology have placed such a means of restraint at our service. Of course it will be said that I advocate the indiscriminate and excessive use of hypnotics in every case of insanity. How far this accusation is justified will be seen from the following remarks:—

In inducing sleep our first object must be to place the patient in those physiological conditions in which sleep normally occurs. What are these conditions? If we knew them all, and could reproduce them, we could induce sleep without administering any drug at all; but, as we know only some of them, this is not always possible, although, since the missing condition may be one of those that we do know of and are able to supply, it sometimes happens that we are able by mere attention to hygiene to induce sleep without administering any drug. The two conditions that we can with the greatest confidence associate with the induction of sleep are fatigue and repletion. Other things being equal, the greater the previous exertion and the greater the fatigue, the greater the tendency to sleep; and not only is there a very definite synchronism between the absorption of food and the induction of sleep, but deficiency of food is inimical to sleep. When therefore we have to deal with a patient who suffers from sleeplessness, it is important to secure for him a healthy degree of fatigue by inducing him to take a healthy amount of muscular exertion, and especially of exertion in the open air; and it is far more important to secure for him the absorption of food. In the majority of cases of acute insanity that come under treatment the patient is suffering from starvation. He has either refused or neglected to take food in sufficient quantity, and his sleeplessness has the same source as that of the man who has had to go to bed supperless. Nothing, not even acute pain, is so efficient a cause of vigil as an empty belly. Even in the torments of toothache and of gout snatches of sleep are possible, but the hungry man does not sleep even for a moment. Hence our first measure in the treatment of acute insanity, accompanied, as it always is, by insomnia, is to give a copious meal of some easily assimilated food. Slops and stimulants, concentrated foods and meat extracts, are not what is needed. What is needed is a bellyful of easily assimilated food;—Benger's food, revalenta, arrowroot, rice and milk, or better, if the patient will take it, and his digestion can cope with it, beefsteak and potatoes, bread and butter—anything, in short, that is both nourishing and bulky. Not seldom this measure and this alone is enough to secure a sound and long sleep to a patient who has scarcely slept at all for weeks. And in any case the treatment is right. The waste of the superior cerebral regions on which the insanity depends may be of such a character that it cannot recover even when copious supplies of pabulum are presented to it; but whatever its character, it certainly cannot be made good without material to make it good; and hence, in all cases of acute and recent insanity, copious feeding is required. Young people sometimes come under care with the history that their appetite is good, that they have been eating pretty well. But in such cases the statement must be received with a qualification. The amount of food that they have taken may have been sufficient for the needs of a mature adult; but the mature adult, the parent who gives us the account, does not realise how greatly the need for food of the adolescent and the immature adult exceeds that of the mature adult. The lad of 18 or 20 needs at least three times as much food as the man of 45 or 50, and what is sufficient, judged by the standard of the one, is very insufficient for the needs of the other. On every account the administration of abundance of food is the first necessity in the treatment of acute insanity, especially in the young.

But it is not enough that food should be administered. It must be absorbed. And in acute insanity, especially in melancholia, the digestive functions are almost always disordered. Even in the sane, the depression that accompanies dyspepsia is sufficiently notorious. In morbid depression dyspepsia, or other disorder of the processes of the gastro-intestinal canal, is scarcely ever absent. This, then, must be the next care of the alienist physician, and here again the value of regulated exercise and a well-ordered diet will soon make itself apparent.

If food alone is insufficient to induce sleep, and commonly it is not by itself sufficient, attention must be paid to the circumstances under which sleep is sought, which may include some that are preventing sleep. Every one knows the greater difficulty of sleeping in the sweltering heat of summer as compared with that of sleeping in cold winter nights; and many a case of insomnia may be relieved by the removal of two or three superfluous blankets. Coldness of the feet is a very effectual preventive of sleep, and the insertion of a hot bottle in the bed will often render the administration of a drug unnecessary. The importance of quiet need not be insisted on, and of all the complaints made by patients of ill-treatment in lunatic asylums, none has seemed to me more bitter, nor better founded, than that which refers to the flashing of a lantern in the eyes of sleepers by the night watchman, a practice which is highly inimical to sleep, and ought not to be permitted.

Supposing, however, that when attention has been paid to all these matters of hygiene, and that the patient is still sleepless, resort must be had to hypnotic drugs, and of these we have a large number to choose from. The particular drug chosen will vary with the character of the malady, but in all cases the best practice is to begin with a large dose, and to give this dose time to act, rather than to give repeated small doses at frequent intervals. When a patient is in a state of very acute mania, raving and raging and ramping, there is no drug that approaches in rapidity and efficiency of action to hyoscin. It is said to be uncertain in its action, and occasionally to produce dangerous effects, but these I have never seen, although I have used it largely, and in much larger doses than are customary; and I am inclined to believe that where it has had ill effects it has either been improperly prepared, or has been administered in larger doses than the administrator was aware of. In such a case as has been mentioned, my practice is to give one-fiftieth of a grain hypodermically or one-twenty-fifth of a grain by the mouth, and usually the patient is asleep in five minutes, and wakes after from six to ten hours without an unpleasant symptom. The effect of the drug, even when administered hypodermically, is sometimes delayed; I have known it to take no effect whatever for as much as two hours; but it must not be repeated for at least twelve hours, and I have never known it to fail. It may be that thus to produce a tranquil sleep almost immediately in a patient who was howling, roaring, dancing, struggling, smashing, biting and kicking—it may be that to produce in him an instant and tranquil sleep, from which he awakes quiet and comparatively rational, or, as has happened in one case under my care, from which he awakes completely and permanently sane—it may be that this is chemical restraint, and if it be, let us be thankful that we have such a means of restraint at our command.

When the mania is less acute and of a more lasting character, and especially in the excitement of general paralysis, no drug has the efficacy of sulphonal, the action of which is somewhat peculiar. It is very insoluble, and has no hypnotic effect for some hours after its introduction into the stomach; on the other hand, its action, unlike that of hyoscin, is very enduring. After a sufficient dose of sulphonal a maniacal patient will not only obtain several hours' sleep, but after he awakes the drug will maintain its action, and the patient will be tranquil, but rather stupid, and apt to be drowsy for the whole of the following day. In other words, as sulphonal is slowly absorbed, so it is slowly eliminated; and when it is given by routine in daily doses it accumulates in the body and produces disastrous effects. Sulphonal should never, therefore, be given continuously. Its use should be alternated with that of other hypnotics, and even when not alternated it should not be given for more than three or four days in succession. In view of the delay in its absorption, it is always wise to combine, at any rate, the first dose of sulphonal with a less quantity of its congener trional, whose action is much more rapid and more transient. The trional puts the patient to sleep, and the sulphonal maintains the sleep. For the first night 30 or 40 grains of sulphonal may be combined with 20 of trional; and this may be followed the next afternoon by 30 grains of sulphonal, and the third or, at any rate, the fourth night, the patient will usually sleep without any drug.

Bromide of potassium, chloral hydrate, and opium with its component alkaloids, are little used as hypnotics in the treatment of the insane. Bromide of potassium has its use as a calmative in the mania which is associated with epilepsy, and when administered for this purpose is of little use in less than drachm doses. Chloral hydrate is not to be recommended except as a variant to be used during the intermission of other drugs. Opium is now perhaps as much neglected as, when our resources were more limited, it was used in excess. As a hypnotic, indeed, it has little value, but it often appears to have a distinct ameliorative effect in melancholia, especially in the melancholia of middle and advanced age, given regularly two or three times a day. Of course in thus administering opium or morphia we run great risk of establishing an opium or morphia habit, but this is a risk that must be run. The choice is between two evils—between leaving the patient in the most wretched condition in which a human being can be, or relieving him at a cost which is indeed great, but which both he and all to whom he is dear would be willing to pay for his relief. As a mere hypnotic the most appropriate drug in melancholia is paraldehyde.

Apart from the administration of food as an hypnotic, it is of very great importance upon other grounds in the treatment of insanity. Less important, perhaps, in the insanities of old age and middle life than in those of youth, it is yet important in all. As most cases of recent and acute insanity that come under care are found to have been for some time sleepless, so most of such cases are found to have been for some time underfed, and a copious supply of food is usually as important an element in their treatment as is the induction of sleep. Often it happens that the patient will not take food; more often that the food that he does take is not assimilated. There are patients who will not eat in company, or if they know that they are observed; and there are patients who will not eat alone. There are those who will not eat from conscientious scruples that they do not deserve it, or that it is not paid for, or that they are not entitled to it; and there are those who will not eat anything that they have not previously stolen. There are patients who take insane prejudices against this or that kind of food, and again patients to whom this or that kind of food alone is acceptable. By far the most important peculiarity is the refusal of food, and this may be of various degrees of intensity. With some patients it is the first step only which costs, and after he has been compelled to take a spoonful he will eat the rest of his meal voluntarily. Another will take nothing voluntarily, but will allow himself to be spoon fed; others will need to be fed with the tube, but will take contentedly whatever is thus administered; while yet others will resist to the uttermost any attempt to feed them, and when at length they are fed will thrust their fingers into their throats and do their utmost to induce vomiting of the food given. Very many methods have been adopted of feeding those who refuse food, and an account of most of them may be found in Hack Tuke's *Dictionary of Psychological Medicine.* Practically the methods may be reduced to three—the spoon, the nasal tube, and the ordinary stomach tube. The spoon should be used where it can be used, and where it cannot the stomach tube is undoubtedly the best alternative. The numerous deaths that have occurred from pneumonia after the use of the nasal tube are alone sufficient to condemn it. A patient who refuses food is suicidal, and the introduction of food by the nasal tube gives him an opportunity of inhaling the food purposely if this do not occur by accident; or if the tube is passed sufficiently far to obviate this danger he can bring it forward into his mouth, bite off the end, and swallow it. In feeding by the stomach tube it is important to remember two precautions. The tube should be well softened in scalding water, and the food should be strained through a sieve so as to free it from lumps which would block the tube. No reliance can be placed upon the assurance of the cook that the food has been strained, and the operator should strain it himself or have a strainer inserted into the funnel. With regard to the nature of the food, the slops enumerated on a previous page are the most appropriate. Liebig's extract, Bovril, Valentine's meat juice, and other extracts can be added if it is thought expedient; but as their nutritive value is low, and as there is no palate to consider, there is not much object in doing so. More important is the administration of helps to digestion, of zymine, of pepsine, lacto-pepsin, etc.

In melancholia, and in acute insanity generally, there is almost always some disorder of function of the stomach and intestines, and this has to be considered and dealt with. Usually when the stomach tube is passed a few bubbles of air will escape from the stomach, and in some cases this air is horribly fœtid. When this is the case the stomach should be systematically washed out before the administration of food, and after its fœtid contents have been withdrawn it should be flooded with a solution of permanganate of potash or, better, of sanitas oil, which has a much more powerful deodorising effect, and to this treatment may be added the administration of β-naphthol or other intestinal disinfectant.

Apart from those that have been enumerated, our means of dealing with the morbid processes that underlie insanity are extremely limited, as indeed may be inferred from our ignorance of their nature. It is found, however, that there are certain morbid states of body which accompany certain forms or cases of insanity, and that the insanity fluctuates, recovers, and relapses, *pari passu*, with the bodily state. The connection of melancholia with gastro-intestinal disorder has already been referred to, and cases have been recorded of cystitis and other bodily maladies with which insanity coexisted and with which it varied. Hence it is incumbent in every case of insanity to investigate thoroughly the bodily health, and to treat any bodily disorder that may be discovered in the hope that with its improvement the mental state also may improve.

The opinion is more and more gaining ground that in a large proportion of cases of insanity the disturbing agent is a poison of some kind. In general paralysis, in acute delirious mania, in puerperal, alcoholic, and saturnine insanities this mode of origin exists without doubt; and true curative treatment of insanity will begin when our knowledge is sufficiently advanced to enable us to eliminate, destroy, or counteract these poisons before they had produced irretrievable structural damage. This knowledge we have not yet reached, and in the meantime our therapeutical measures are limited to treating whatever morbid process we can find that is amenable to treatment, and so, by inducing a general state of good health, to place the tissues of the individual in the most favourable state for eliminating or destroying the poison.

In view of this mode of the causation of insanity, it is upon organotherapy and serum-therapy that our hopes are chiefly based of eventually treating insanity in a scientific manner, and the tentative efforts that have been made in this direction are not destitute of encouragement, though more than this cannot yet be said of them. The administration of thyroid extract has been tried in a large number of cases besides those of cretinism and myxœdema, in which its action is so marvellous, and in a few of these cases recovery has followed under circumstances that leave little doubt that the recovery was due to the administration of the extract; but no class of cases has yet been identified as specially amenable to this mode of treatment, and treatment by serum-therapy has not yet begun.

While many of the poisons that serve in the production of insanity are unknown, and while those that are known are not amenable to the action of any known antidote, there are some which, if we cannot deal with them when once they have gained entrance to the body, we can at least arrest at the door and prevent from entering. We cannot undo the mischief that alcohol has done to the brain, but we can at least see to it that no more alcohol is imbibed, and so of lead, morphia, chloral, and other ingested poisons. In puerperal insanity, also, we can clear out the uterus, and counteract the septicity of its contents, which, in some cases at least, appears to be contributory to the insanity.

Dr. Haig has written much and strenuously upon the evil effect of an animal diet in bringing about uric-acidæmia and melancholia, and upon the advantages in this psychosis of regulating the diet and administering salicylates. There is no doubt that in melancholia the blood-pressure is high, but beyond this there is nothing in the majority of cases of melancholia to corroborate Dr. Haig's doctrine. It is very rarely that a case of melancholia is improved by the administration of salicylates, but here and there we do meet with a case, usually that of a portly, middle-aged man who has lived pretty freely, whose depression does appear to be favourably influenced by the administration of these compounds. The cases in which they are likely to be useful are those in which the urine, scanty and of high specific gravity during the day, is copious and watery in the night, and in which the patient has to get out of bed once or twice to pass water —cases, in short, of incipient renal disease. The combination of depression of spirits with these symptoms is not very common, in spite of all that Dr. Haig maintains, but when they are so combined the administration of salicylates has a very marked effect in improving both the one and the other.

There are certain phases of conduct exhibited by the insane which demand special notice with reference to their treatment.

Suicidal Tendency.—This is extremely common in acute insanity. There is a prevalent opinion among those whose experience of insanity is limited that mania and melancholia are entirely distinct forms of the malady, and that the tendency or impulsion to suicide occurs in the latter form only. This view is entirely erroneous. It is true that the deliberate intention to commit suicide is very often associated with melancholia, but it is also true that conduct of the most determined and persistent suicidal character is often exhibited in states of acute mania in which no other sign of depression is apparent, and in which there does not seem to be enough mind for the formation of deliberate intention. It is in such cases that refusal of food is most obstinate, and that impulsive rushes to the windows, attempts at strangulation, and so forth, are most common. For the prevention of suicide in these, as in other cases, there is but one method which has any chance of success, and that is the exercise of incessant vigilance. Under no circumstances, neither by night nor day, must the patient be left unwatched for a single moment. He must be accompanied in his goings out and his comings in. He must be taken to the closet and watched while there, and an attendant must sit by his bedside all night. And the attendant must be experienced in the ways of such patients, and fully impressed with the necessity of making the vigilance incessant. A patient whose head is under the bed-clothes is practically as well screened from observation as if he were in a room alone, and in such circumstances can stuff his mouth with a corner of the sheet, and so suffocate himself effectually, while his attendant sits by unsuspecting.

Masturbation is a very common habit in the insane of both sexes, and one that is very difficult to counteract. Those who are still sufficiently governed by the restraint of modesty to restrain from it while they are under observation should be kept under observation to the same extent as a suicidal patient, but many of those who are addicted to this habit own no such restraint. In them continuous and laborious employment is the most effectual remedy. They should be worked in the garden, the field, or the shop until they are dog-tired, kept up late, made to rise early, and lightly clothed at night. In those cases in which the habit is most fixed, the patients are as a rule too demented to be capable of any but the simplest employments, but still they can be harnessed with others to a cart or a roller, and thus kept from the indulgence. When they are not physically fit even for this mode of exercise their dress can be so arranged that their genitals are inaccessible to them, but this expedient is far less successful.

Faulty habits as they are called, that is to say, the emptying of the bladder and rectum into the clothes, are very frequent among the insane, and are due, sometimes to the mere negligence of dementia, sometimes to the desire to cause trouble and annoyance, and sometimes to a strange but undoubted association between the performance of these acts and the satisfaction of sexual desire. In most cases the fault can be overcome by observance of the routine of visiting the closet at moderately frequent intervals, that is to say, after every meal, and once or twice during the night.

Destructiveness is sometimes an impulsive outbreak of Berserker rage, of which no explanation can be given, and against which scarcely any precautions are adequate; but more often it is the resource of vacuity. A man has a certain amount of energy to expend, and this energy will find egress in some mode of activity. If no useful mode of activity presents itself or is available, it will find egress in twisting the buttons off his coat, in tearing his clothes, in pulling things to pieces, and so forth. The remedy is obvious. He should be found something to do. If he is too demented to undertake any more intelligent occupation than that of tearing, he should be given newspapers to tear.

Mechanical restraint is practically never used in institutions specially devoted to the treatment of insanity, although the imbecile wards of workhouses are not considered properly equipped without a supply of strait waistcoats and of buckled sheets for fastening troublesome patients down in bed. While restraint to the extent which this provision of appliances implies is never required, and is improper, it is a question whether the total abolition of mechanical restraint is not an unnecessary and an undesirable sacrifice to convention and sentiment. Certainly mechanical restraint should never be employed, and should be rendered illegal outside of lunatic asylums; but with the usual logic of that law, which its professors are fond of describing as the perfection of common sense, the use of mechanical restraint, in the last resort, in places in which its use is surrounded by every imaginable safeguard, is pounced upon, discountenanced, and considered disreputable; while its use by persons who are very incompetent judges of its necessity or desirability as a frequent means of control in cases in which it is neither necessary nor desirable, is not forbidden in private houses or in workhouses.

While the cases in which the use of mechanical restraint is permissible are undoubtedly very few, there is a small residue in which it is sometimes of service and even very beneficial. A patient will sometimes beg to be tied down or put in a straight waistcoat in order to prevent him from giving way to his impulse to injure himself or others; and in such a case the feeling of confidence and peace that is engendered by the restraint is far greater than can be attained by the knowledge of watchfulness on the part of his attendants, and is well worth the sacrifice of a principle which, carried to excess, is but a sentiment. Cases of very persistent masturbation are sometimes impossible to control by any but mechanical means, and in such cases the mechanical restraint is certainly a less evil than continual struggling and interference. The same may be said of cases of very persistent self-injury. In this, as in other matters, there is a degree of fanaticism which is unjustifiable and pernicious. While it is undoubted that the use of mechanical restraint should be minimised to the utmost, it is a mistake to suppose that it would be an unmixed benefit to abolish it altogether.

LITERATURE.—1. *The Journal of Mental Science* should be consulted for detailed accounts of recent progress in Therapeutics, etc. 2. HACK TUKE's *Dictionary of Psychological Medicine*. 3. *Text Books*, CLOUSTON, CAMPBELL, CLARK, MACPHERSON, BEVAN LEWIS. 4. For literature dealing with Pathology, see p. 321.

Insects, Diseases due to. *See* MYIASIS.

Insolation. *See* SIRIASIS.

Insomnia. *See* HYPNOTICS, SLEEP.

Insular Sclerosis. *See* PARALYSIS.

Intertrigo. *See* ERYTHEMA.

Intestines, Diseases of (Medical).

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See also Colon, Gastro-Intestinal Diseases,
Indigestion, Melæna, Sand.

Anatomical and Physiological Considerations.—It is of importance in the study of diseases of the intestine to have a clear knowledge of the development of the intestinal tract. At an early stage of the human embryo, while the three layers of the epi-, meso-, and hypoblast still remain distinct, and the neural canal and notochord are in the process of formation, the forward growth and over-folding of the mesoblastic layers immediately in front of the anterior end of the neural groove, form the head of the embryo in which the blind fore gut appears, while at a slightly later period a protrusion from the posterior blind end of the groove curves downwards and forwards to form the hind gut.

Coincident with the formation of the fore and hind gut, the lateral sulci, as they dip down, mark off the body cavity of the embryo from the rest of the blastoderm to form the middle part of the alimentary canal, which for a time remains open, and then gradually becomes pinched off from the yolk sac. Later, by invagination of the epiblast anteriorly and posteriorly, the mouth and anus are formed.

The further development of the pharynx, œsophagus, and stomach continue, while the intestine still retains a connection with the yolk sac by its duct. This gradually becomes constricted by ingrowth from the sides, remaining for some time as the omphalo-mesenteric or vitelline duct, the remains of which in after-life are represented by the permanent cicatrix known as the umbilicus. It thus happens that the whole of the intestine, with the exception of the invaginations of epiblastic tissue from without to form the mouth and pharynx and anus, is formed from hypoblast. The organs of secretion which discharge their contents into the intestine (liver, gall-bladder and its biliary ducts, pancreas, gastric and intestinal glands, etc.) are developed round tubular extensions from the intestinal tract, and are also hypoblastic structures, with their excretory ducts lined by epithelium of hypoblastic origin. The mesentery and peritoneum are developed from mesoblast. It is of interest in this connection to note that most primary malignant diseases of the intestine commence in the glandular structures, and are of hypoblastic origin and of a cancerous nature, and that primary tumours of the peritoneum are sarcomatous in structure.

Anatomically the intestine, about 26 feet in length, is divided into large and small, the former being one-fifth the length of the latter, but in diameter twice as great. The small intestine is arbitrarily divided into three portions, the first 10 or 12 inches, below the pylorus, being known as the duodenum, the upper two-fifths of the remainder the jejunum, and the lower three-fifths the ileum. The duodenum is, except when immediately adjacent to the pylorus, practically fixed, while the jejunum and ileum are freely movable by virtue of their being possessed of a mesentery. The large intestine, commencing at the lower end of the ileum, is divided into the cæcum, to which the rudimentary vermiform appendix is connected, the ascending, transverse and descending colon, the sigmoid flexure, and the rectum. The cæcum is that part of the large intestine which, blind at its lower end, lies below the level of the ileo-cæcal valve, and, unlike any other part of the intestine, is entirely covered by peritoneum. The sigmoid, the ascending and the descending colon have normally no mesentery, and are more or less fixed, but the sigmoid flexure, though usually situated in the left iliac region, is capable of extensive movement within the abdominal cavity, a fact of considerable clinical importance. The structure of the small intestine, speaking generally, is the same throughout its length. It has a serous coat, a muscular, a submucous and a mucous layer. The serous coat entirely surrounds the intestine, except at the mesenteric attachment. The duodenum is but partly surrounded, and the mesentery of the colon, and upper part of the rectum, contains numerous little projections enclosing fat, and known as appendices epiploicæ. The muscular coat is disposed in two layers, an external longitudinal and an internal circular. In the small intestine the disposition of the longitudinal coat is uniform, but in the large intestine it is so arranged as to constitute three well-marked longitudinal bands. The submucous coat is a layer of areolar tissue between the muscular and mucous coats. The mucous membrane is lined throughout with columnar epithelium, and contains the various secreting and absorbing structures necessary to digestion and absorption. Its superficial area is greatly increased by the presence, from immediately below the opening of the bile and pancreatic duct, of the valvulæ conniventes. These are very numerous, high in the jejunum, but fade gradually until they disappear towards the lower part of the ileum. The intestinal villi on and between the valvulæ conniventes are in structure similar to the mucous membrane itself. They are covered with columnar epithelium, and below this a basement membrane is found. In their structure also are included the lacteals, blood-vessels, and nerves, which are supported by a loose network of lymphoid tissue, with a few muscular fibres. The glands of the mucous membrane are Lieberkühn's, Brunner's, Peyer's patches, and solitary glands, which are known in the large intestine as lymphoid follicles. Brunner's glands occur in the duodenum only, and are branched tubular glands identical with the pyloric glands of the stomach. They lie in the submucous coat, and their ducts pierce the mucosa. Lieberkühn's glands are simply tubular or test-tube like glands. They occur throughout the length of the intestine, but are more numerous in the large than in the small intestine. They are lined with a low, cylindrical epithelium between which are numerous goblet cells, these being more numerous in the glands of the large intestine. Lieberkühn's, and to a less extent Brunner's glands, secrete the succus entericus. The solitary glands are found throughout the large and small intestine, and in all parts of their circumference. They are the size of millet seeds, and consist of lymphoid tissue. Peyer's patches are collections of the above lymphoid nodules arranged together in groups, of an oblong outline. They vary in length from one to three or four inches, are most numerous in the lower part of the ileum, are not found below the ileo-cæcal valve, and rarely occur as high as the upper part of the jejunum. They are situated opposite to the mesenteric attachment, and their long axis corresponds to the long axis of the intestine. These patches are best marked in youth, and after middle age begin to atrophy, and in old age are seen frequently only as pigmentary discolorations of the mucous membrane. Typhoid ulcers are the result of their destruction. The arterial blood-supply to the intestine is for the most part derived from the mesenteric arteries, but the duodenum is also supplied by several other branches, notably from the gastro-duodenal. The branches of the mesenteric artery run transversely below the serous coat, then perforate the muscular coat, and form a close network in the submucosa, which gives off branches freely to the vascular mucous membrane. Each villus is supplied by an artery which enters it excentrically. In the small intestine the veins accompany the arteries, but in the large intestine the arteries form a meshwork in the mucous membrane, and the veins return the blood in a manner similar to the blood-vessels of the mucous membrane of the stomach. Anatomically the anastomosis in the intestine is very free. In disease it would appear that under certain conditions the plugging of even a considerable branch by, say, a cancerous growth invading the vessel, can produce an annular gangrene of the intestine. The lymphatics or lacteals of the intestine commence as the lymph vessel or space in each villus. These unite with other vessels to freely provide the mucous membrane with a form of plexus in the submucous coat. Another plexus exists between the muscular coats, and these two uniting with the lymphatics under the serous coats on the mesenteric border form larger vessels, which empty themselves into the mesenteric glands, which again discharge their contents into the thoracic duct. The nerves of the intestine are derived from the solar plexus, and more particularly from the superior and inferior mesenteric plexus. The nerves accompany the arteries, perforate the muscular coat, and between the two layers form Aurbach's plexus, and in the submucous coat Meissner's plexus. Narrow filaments from Aurbach's plexus supply the muscular coat, and, from Meissner's plexus filaments pass into the mucous membrane and the villi, but their ultimate termination is doubtful. The solar plexus from which the nerve supply of the intestine is derived is formed by the great semi-lunar ganglia which receive branches from the splanchnics and vagus. Stimulation of the vagus nerve increases peristalsis, and stimulation of the splanchnics inhibits movement if the blood in the intestinal vessels is normal, but if the blood in the intestinal capillaries is venous, the reverse effect occurs. The splanchnic is in addition the vaso-motor nerve of the intestine, and controls the largest vascular area in the body. It is also the afferent sensory nerve. Under Digestion and Metabolism the physiological functions of the various structures above enumerated in relation to secretion and digestion of fluids, and absorption of digestive products, are discussed. There the digestion of proteids, carbohydrates, fats, etc., by the various juices and their enzymes, with their subsequent absorption by the vessels, lacteals, etc., of the small and large intestine, is described. Intestinal digestion is most active in the small intestine, unless perhaps the greater activity of bacteria in the large diminishes the difference. Recent researches on absorption in the large intestine are of some importance clinically. Vaughan Harley's recent work on this subject deserves notice. Among other conclusions based on the experimental removal of the large intestine, the following occur:—

(*a*) Carbohydrates are absorbed equally well with or without the presence of the large intestine.

(*b*) Proteids are markedly influenced, 10 per cent less absorption occurring when the large intestine is removed.

(*c*) Fats are split up and absorbed equally well with or without the large intestine.

(*d*) The fæces are greatly increased in quantity (five times). This is principally due to the increased quantity of water, that is to say, a large quantity of water is absorbed in the large intestine.

(*e*) Urobilin is not discovered in the fæces after the large intestine is removed. Urobilin is the product of the splitting up of the bile pigments in the large intestine by putrefactive action, probably chiefly bacterial. Under normal conditions urobilin exists in the large intestine, the fæces, and the urine, and is recognised by giving a deep pink reaction with a concentrated solution of perchloride of mercury. Excessive increase in the urine and fæces indicates either an abnormal secretion of bile, or increased putrefactive action in the intestine. Thus we expect to find in diarrhœa of a septic nature an increase of this substance in the urine.

Bacteria of the Intestinal Tract

Before birth the intestinal tract is sterile. The meconium for three to seven hours is free from bacteria, or at the most a few cocci are obtainable. In a few hours bacteria make their appearance in abundance, and throughout life the intestinal tract from mouth to anus abounds in innumerable varieties. It may be said at once that attempts to isolate and classify the bacteria found in the intestine are most incomplete, for the task is herculean.

The Rôle of Bacteria in Health.—Bacteria or spores are introduced into the stomach at every act of deglutition. Many are destroyed, some are aerobic, and by being deprived of oxygen, die; others are destroyed by the hydrochloric acid of the stomach, which is a germicide, while others find no suitable pabulum for their nutrition and growth. By the germicidal action of the hydrochloric acid of the gastric juice the formation of decomposing products and other disturbances produced in the normal course of digestion is prevented. The comma bacillus of cholera is readily destroyed by the gastric juice, as is also the bacillus of splenic fever, though its spores are unaffected. Many pathogenic bacteria are unharmed by the gastric juice, *e.g.* tubercle bacilli. It is a noteworthy fact that the average amount of hydrochloric acid found in the gastric juice practically coincides with the amount required to kill experimentally most fermentative and many pathogenic bacteria.

The Action of Bacteria on Digestion.—In the normal state of the intestinal tract bacteria aid the various secretions and their enzymes, but to what extent is doubtful. Growth and normal nutrition can go on without the presence or assistance of bacteria, as has been proved experimentally by placing newly-born animals in sterile media and feeding them on sterilised food, when they grow and thrive as satisfactorily as under their usual conditions. In the stomach, on account of the action of hydrochloric acid, little digestion by bacterial action takes place. In the early stages of gastric digestion, before the hydrochloric acid is fully active, carbohydrates undergo, to a slight extent, lactic acid fermentation, but proteids and fats are not attacked to any extent, if at all. In the intestine the bacterial action is more vigorous. The less acid reaction of the intestinal contents favours this, but it is also supposed that organic acids are set free by the action of bacteria on carbohydrates, and that these acids to some extent inhibit bacterial action on the putrefaction of proteids. A condition favourable to their activity and one antagonistic appear therefore to go on side by side. Proteids in the intestine are first slowly dissolved and converted into albuminoses and peptone; then these along with the tyrosin of tryptic digestion are broken up into various aromatic compounds, such as indol, skatol, and skato-carbonic acid by bacterial action. Carbohydrates are affected earlier than proteids. Starch is practically decomposed into sugar, and sugar already converted is further decomposed into lactic, butyric, and succinic acids. What change is affected on cellulose during digestion is entirely by bacterial action, or, otherwise stated, bacteria are the only agents which alter cellulose. On fats under normal conditions little action is produced by bacterial action, but in the absence of bile or pancreatic juice they are converted into fatty acids, which pass out of the body unabsorbed in the fæces.

The Rôle of Bacteria in Disease.—Pathogenic bacteria may be introduced into the intestine and directly cause specific disease (typhoid, tubercle, etc.) or those already existing in the intestine may, under altered conditions of nutrition, multiply and be converted from saprophytic into pathogenic organisms. The exact conditions under which saprophytic bacteria become pathogenic are very imperfectly understood, but it would seem as if a simple abrasion of the mucous membrane, the presence of enteritis, defective circulation, local or more general, and even a lowered condition of the body, is sufficient to allow of this change. As illustrations we may mention that in some cases of appendicitis the inflammation is produced by the entrance of the bacillus coli into the mucous membrane eroded by an enterolith or other cause; or cases of uncomplicated enteritis from a mechanical irritant are followed by a putrid diarrhœa of bacterial origin. The intestinal wall in its healthy state effectively resists the passage of bacteria through it, but in certain diseased conditions, as, for example, gangrene following strangulated hernia, peritonitis, etc., bacteria are able to pass through the walls, and cause definite lesions in the intestinal wall itself and in the adjacent peritoneum. The pathological changes following may result either by direct action of the bacteria themselves or by their toxins.

In the intestinal digestion of proteids the putrefactive bacilli which produce poisonous nitrogenous and alkaloid bases known as ptomaines do not flourish under normal conditions, and consequently none are normally present in the intestine. They may be, however, introduced through the medium of tainted meat, either as bacteria capable of elaborating these bases, or as ptomaines already elaborated.

Authorities are not in agreement as to the period of production of ptomaines in the process of putrefaction, some holding that they are late, others that they are early products. If a single variety of intestinal bacterium becomes abnormally active, it is probable that others, on account of the altered conditions produced, will become pathogenic also.

A few of the more important bacteria found in the intestine deserve special attention. 1. Bacterium coli communis is present in both the small and large intes tine in health, and to it are ascribed many of the useful physiological functions of intestinal bacteria. Under normal conditions it is saprophytic, but under altered conditions of nutrition of the intestine or perhaps of the body generally, it becomes pathogenic and plays an important part in the causation if it is not the sole cause of many intestinal diseases. In most inflammatory and suppurative conditions of the intestine it is found in excess. In suppuration of the gall-bladder and bile ducts it has a most important place, also in some varieties of enteritis, in appendicitis, and in peritonitis, whether the intestine is perforated or not, as it can pass through a damaged intestinal wall and produce a distinct variety of peritonitis. In typhoid fever the colon bacillus increases enormously, especially in the large intestine, and its resemblance to the typhoid bacillus requires special cultivations on various media and discriminating reactions to distinguish one from the other.

2. Staphylococci are found in small numbers in the normal intestine, but in catarrh of the mucous surface, and in suppurative and gangrenous conditions and other diseases, they multiply vigorously.

3. Streptococci, which normally exist in the bowel, are virulent in diseased conditions and abound in all inflammations with fibrous exudations, *e.g.* non-diphtheric inflammations, enteritis, and colitis, etc. It is stated that streptococcus longus in the milk of cows suffering from mastitis produces diarrhœa in children, and when experimentally injected into animals.

4. *Bacillus Enteritis Sporagenes.*—Klein isolated this anaerobic bacillus in epidemic diarrhœa, of which milk was supposed to be the cause. His own and further investigations by others have not confirmed this view, but rather go to show that the bacillus exists widely in nature, in the normal intestine, in normal dejecta, in dysentery, diarrhœic stools, ulcerative colitis, etc. Gautner in 1888 isolated a bacillus which he calls bacillus enteritis in some cases of meat poisoning, and another bacillus evidently of the same group has lately been isolated in cases of meat poisoning. Both caused intense hæmorrhagic enteritis.

5. Bacillus pyocyaneus is said to cause gastro-intestinal catarrh, and to be found in the intestine in cases of infantile atrophy.

6. The bacilli of typhoid fever are found chiefly in Peyer's patches and the solitary glands before the stage of necrosis and suppuration, when they to a great extent disappear, remaining at the edge of the typhoid ulcer and in the lymphatic vessels and the mesenteric glands. It is probable that the necrosis is chiefly brought about by the action of the toxic products of this bacillus. As already mentioned the colon bacillus is found in great abundance in combination with the typhoid bacillus.

7. Bacillus of diphtheria is found whenever a true pathogenic membrane forms in the intestine, and where the diphtheric membrane from the fauces is swallowed. It is by no means the only bacterium present in diphtheric membranes, but is associated with streptococci chiefly.

8. Cholerus spirillum (*vide* "Cholera," vol. ii.).

9. Tubercle bacilli may be present in the intestine and fæces in the absence of tuberculosis, yet their presence in the fæces in the absence of lung disease is most suggestive, especially in suspected cases of primary tuberculosis of the intestine in children (*vide* "Tuberculosis and Intestinal Tubercular Ulceration").

10. *Actinomycosis.*—For a description of the actinomycoses or ray fungus in the intestine *vide* "Actinomycosis and Appendicitis."

MALFORMATIONS

1. *Of the Duodenum.*—Occur as duodenal pouches or occlusion.

Duodenal occlusion is met with just above the entrance of the bile duct into the duodenum. It may occur as a complete occlusion, the first portion narrowing gradually so as to present a blind end ununited to the second portion, except, possibly, by a fibrous band, or as a diaphragm complete or perforated, and consisting of structures similar to those composing the intestinal walls. A suggested method of production of both of these conditions is that a kink is produced above the bud when the hepatic diverticulum buds off from the duodenum, and adhesion produces permanent occlusion. The symptoms produced are vomiting, which occurs soon after birth, and the vomited matter is not bile-stained. Jaundice is usually absent, muconeum is passed, and the bowels may be moved several times after birth. Rapid emaciation follows and death takes place in from one to eleven days, the time varying with the degree of obstruction.

Duodenal diverticula or pouches occur in the same situation as occlusion, namely, just above the biliary papilla. The pouches are wide-mouthed, and their walls consist of the same structures as the intestinal walls. As a rule they do not attain to any great size, and are not known to give rise to any symptoms unless, as has been occasionally reported, gallstones or other foreign bodies become arrested in them. These diverticula appear to be connected with the hepatic offshoot in much the same way as Meckel's diverticulum is with the vitelline duct. They appear to be related to an erratic form of developmental activity at this portion of the intestine. The duodenum has also been found unattached to or occluded near the pylorus at its upper end, and a similar defect has been noticed near the jejunum at its lower end.

2. *Of the Small Intestine.*—(*a*) *Meckel's diverticulum.*—In early fœtal life the intestine communicates with the yolk sac by the vitelline canal, and a persistence of this communication may leave a tubular connection between the umbilicus and the intestine. This may be occluded at any part of its course, and this remnant of fœtal life (met with in about 2 per cent of bodies examined) is known as Meckel's diverticulum. If the tube remains open at the umbilicus, it forms one variety of fæcal fistula (*vide* "Intestinal Obstruction").

(*b*) *Multiple diverticula* of the small intestine are said to have their origin in protrusions from within, the result of defective resistance of limited areas of the intestinal muscular coat, or dragging from without, the result of adhesions due to congenital peritonitis.

(*c*) *Occlusion of the small intestine* by diaphragms stretching across the lumen occur singly as a rule, but are occasionally mutiple. Their etiology has received no satisfactory explanation, but it has been suggested that they resemble the similar malformations in the duodenum, and arise from excess of the atrophic process which results in the obliteration of the vitelline duct. It is also possible that intra-uterine peritonitis may, by the formation of bands, produce the same result. Occlusion of the small intestine is incompatible with more than a few days of life, but as distinguishing symptoms from duodenal occlusion vomiting of biliary matter and abdominal distension will usually be recognised, the latter in proportion to the distance of the obstruction from the stomach (see also "Gastro-Intestinal Disorders of Infancy," vol. iv.).

(For malformations of the colon *vide* "Colon," vol. ii.)

Enteritis

Definition.—In its widest sense enteritis implies inflammation of any part of the intestine large or small. This generic definition is, however, by common usage, restricted to inflammation of the small intestine. The corresponding inflammation of the large intestine is known as colitis, and is described under that heading.

Etiology.—Enteritis is caused by contact of an irritant of sufficient virulence with the intestinal wall. It may also be produced by an extension of inflammation from other neighbouring structures or organs, and it is then known as secondary enteritis.

Predisposing Causes.—(*a*) *Debility.*—Feeble and reduced health, particularly in very old or very young people, predisposes to enteritis.

(*b*) *Age.*—Children suffer far more frequently than do adults. Indeed a mild catarrhal enteritis is such a frequent accompaniment of the period of lactation as to make it probable that the highly active digestive tract of early childhood is specially susceptible to inflammation.

(*c*) *Season and Temperature.*—Atmospheric conditions and environment exercise a marked effect upon the prevalence of epidemic enteritis commonly known as summer diarrhœa. The third quarter of the year, July, August, and September, has double the number of deaths from this cause that the combined remaining nine months of the year have. In a dry, hot summer and autumn more cases of epidemic enteritis occur than in a cool wet season. It is more prevalent in the ill-ventilated and dirty streets of large towns than in more healthy localities, or in the country. The facilities for its spread are greater in crowded localities, but dirt, the want of sunshine, and fresh air among the children, and other circumstances favourable to bacterial growth, errors of feeding, spoiled food, and milk, etc., are all powerful factors in favour of its occurrence.

(*d*) *Exposure to cold* has the same relation to enteritis as it has to pleurisy or nephritis, but another factor, namely, disordered digestion by producing an intestinal irritation, may act as a more direct exciting cause.

Exciting Causes.—These may be mechanical, chemical, or bacterial. Of mechanical causes the most potent are indigestible and undigested food. For instance, the firm coagulum of milk may cause diarrhœa in children, unripe fruit too freely eaten, fruit containing much woody fibre, nuts, fruit-stones, gallstones, intestinal worms, etc. Among chemical irritants capable of causing enteritis may be mentioned violent purgatives such as croton oil; other irritants include the caustic alkalies, carbolic acid, arsenic, corrosive sublimate, etc. Under this heading also may be placed the unascertained irritant which causes enteritis in some cases of extensive superficial burns.

Bacterial causes of enteritis assume a more and more important place as our knowledge increases. The exact method of action of the micro-organisms is as yet imperfectly understood, but it is now generally recognised that the bacteria normally existing in the intestine may, under certain altered states of nutrition of the individual, of the intestine itself, or of its contents, become pathogenic, or that one species of bacterium, becoming abnormally active may so alter the normal equilibrium that other species can multiply and become virulent; or bacteria introduced into the body and coming in contact with the intestine may cause inflammation.

In the former class probably the best example is furnished by the bacterium coli communis, which, normally saprophytic and useful under healthy conditions, may become under abnormal conditions pathogenic, as, for example, above the site of a strangulation, when the resulting peritonitis is due to this organism becoming unusually virulent and invading the intestine after its vitality and nutrition have been lowered by strangulation.

In the second class, bacteria introduced into the stomach and intestine from without, the medium is almost always some form of foodstuff which is more or less decomposed, as, for example, tinned, preserved, and potted meat, tinned fish, putrefying meat, pork more frequently than other kinds, sour milk, high game, oysters, etc. In this class of food poisoning the poison may be either bacterial or the product of their action, which results in the formation of ptomaines, and the symptoms produced are known as ptomaine-poisoning (*vide* "Food," vol. iii.). There is every reason to believe that this is due to a poisonous animal alkaloid formed, it is generally supposed, in the later stages of putrefaction and not to bacteria themselves. The exact alkaloid has not yet been isolated, but a substance called "sepsin," capable of producing intense hæmorrhagic enteritis, has been obtained by Bergmann from decomposing yeast, and Briegar has secured nuerine, coaline, and murodine, which closely resembles the poison of mushrooms—muscarine—from decomposing meat. Alkaloids, resembling curara in their action, have also been separated from decomposing fish. All of the alkaloids thus separated are not poisonous, but some are extremely toxic. All toxic ones cause enteritis, cardiac failure, and paralysis or convulsions.

In epidemic enteritis (summer diarrhœa of children) it is probable that no one bacterium but many are the potent factors in its causation. Attempts have been made to classify cases according to the bacteria found in the stools, but such a classification is, for the present at least, impracticable. Klein separated from the stools in an outbreak of enteritis a bacillus which he called bacillus enteriditis sporagenes, but he and others, by subsequent investigations, have failed to prove this anaerobic organism to be the specific bacterium of this disease.

A break in continuity of the intestinal mucous membrane favours the entrance of bacterium coli, and enteritis, with subsequent ulceration, is likely to supervene, as exemplified in some cases of traumatic appendicitis, or intestinal cancer or intestinal obstruction when the intestine at and above the lesion is frequently inflamed and ulcerated, and may be gangrenous as a result of bacterial infection of the damaged intestinal walls (*vide post* "Bacteria of the Intestine").

Pathological Anatomy.—The superficial mucous membrane is in mild cases the site of inflammation, but in more severe forms inflammation extends to the submucous coat. The initial lesion appears as a diffuse redness of the mucous membrane, and the exudation of inflammatory products causes œdema and tumefaction. The surface of the inflamed mucous membrane is usually coated with a tenacious layer of mucus, often blood-stained. Lieberkühn's follicles, Peyer's patches, and the solitary glands are swollen and prominent; their ducts become obliterated, small erosions or catarrhal ulcers develop, and, if the inflammation is severe, hæmorrhage into and under the mucous membrane takes place. Unless the case becomes chronic or the inflammation be very acute, the muscular and serous coats are slightly, if at all, affected. If, however, the case becomes chronic, the inflammatory products develop into new connective tissue, the glandular elements are to a great extent destroyed, the muscular and serous coats become involved, the intestinal wall is infiltrated by the products of chronic inflammation, the mucous membrane at the same time shows considerable erosion, and by sloughing of the glandular elements, consequent on tension produced by plugging of their ducts, catarrhal ulcers are formed. The secretive and muscular functions become then permanently damaged. In certain cases further mischief may follow; localised abscesses may form and break into the bowel, or gangrene, with perforations of the bowel, may result.

Enteritis rarely affects any considerable length of the small intestine; usually a few yards only are involved. The colon is more frequently affected than other regions, and the jejunum probably less frequently than any other part.

Symptoms.—The symptoms of acute intestinal catarrh vary greatly in severity, and to a large extent in accordance with the cause. An acute irritant, like carbolic acid or a caustic alkali, will produce a more acute diarrhœa than that due to a simple mechanical irritant. As a rule, the earliest symptom, as in most febrile affections, is chilliness. A feeling of uneasiness in the abdomen, followed by colicky pains, nausea, and loss of appetite, are complained of early. The nausea may be followed by vomiting and hiccough. The tongue is dry and coated, the expression is anxious, and the face pinched; thirst is complained of, diarrhœa soon commences, the stools being at first semi-solid and gruel-like, but later watery, acid, and scalding, containing more or less mucus in accordance with the site of the enteritis; the higher in the intestine the less the mucus in the stools. (A stool almost wholly composed of mucus may with a fair degree of certainty be said to have its origin below the splenic flexure of the colon, *vide* "Fæces," vol. iii.) The stools are usually numerous, and often become fœtid, either from decomposition in the intestine by bacterial action or from temporary arrest of the biliary secretion. The colour may be green from biliverdin staining, especially in children, but as a rule the evacuations remain clear and watery, consisting of unabsorbed intestinal contents—epithelium, mucus, food particles, leucocytes, other products of inflammation, and innumerable bacteria, of which the bacterium coli spores and rod-shaped bacteria are the chief. In proportion to the amount of fermentation going on in the intestine there will be abdominal distension, borborygmi and increased flatus passed per anum, and palpitation and cardiac discomfort may arise from upward displacement of the diaphragm. Diarrhœa may be absent in inflammation of the duodenum or of the upper part of the jejunum, the remaining healthy intestine below absorbing the surplus fluid before it reaches the rectum. In children and old persons more particularly emaciation may be rapid, and strength quickly lost, especially if the diarrhœa be profuse. Urine is scanty and high-coloured. The temperature is variable, but as a rule is not high. Pulmonary œdema and cardiac failure in severe cases complicate the disease, and occasionally epileptiform convulsions supervene towards the end. In accordance with the cause of the enteritis the symptoms necessarily vary.

The symptoms of ptomaine-poisoning call for special notice. At a variable interval, an hour or two, or as long as thirty-six hours, after eating tinned salmon, high game, etc. the patient complains of a feeling of nausea, restlessness, and sickness. This is speedily followed by vomiting and gradually increasing abdominal pain, which soon becomes agonising. Muscular cramps and clonic spasms, especially in the legs, add to the patient's misery, and soon a feeling of intense prostration, accompanied by cold sweats, nervous depression, and cardiac failure, appears. The expression is anxious and even terrified; diarrhœa usually supervenes early, is profuse, watery, often fœtid, and sometimes bloody. Intense thirst is nearly always complained of, the pulse is quick and small, the urine is scanty, and the temperature varies from 100° to 103°. An urticarial or erythematous rash is not uncommon. If the dose of the poison has been large, and especially if diarrhœa does not early carry off some of the poison, the patient may lapse into a state of unconsciousness and coma. The pulse becomes quicker and more feeble, and cardiac inhibition becomes gradually more marked before death.

The severity of the symptoms varies not only with the amount of the poisonous dose taken or elaborated after ingestion, but different individuals who have partaken of the same meal and in similar quantities are not equally affected. Assuming that the poison is equally distributed in the food, personal idiosyncrasy must enter into the calculation; but there are many fallacies.

The symptoms begin to abate at a variable period of a few hours or days, but in any case convalescence is slow.

Epidemic enteritis of children, otherwise called summer diarrhœa, or septic diarrhœa, is characterised by the sudden onset of vomiting and diarrhœa. The stools—at first frequent, offensive, and solid—later consist of watery serum, mixed with flaky mucus. This disease is further characterised, on account of the rapid loss of fluid through diarrhœa and vomiting, by rapid emaciation, intense thirst, and collapse, which may in a single day result in death (*vide* "Gastro-intestinal Diseases of Infancy and Cholera Nostras").

Diagnosis.—A history of improper feeding, followed by colic, nausea, diarrhœa, rise of temperature, and quickened pulse, indicates the nature of the case. It is, however, often very difficult to determine the part of the bowel affected. Catarrh of the duodenum may be accompanied by jaundice, and often is by gastric catarrh; or pain localised in the right hypochondriac region may suggest the site. The diagnosis of inflammation of the jejunum from that of the ileum is generally impossible. The distinctive points between inflammation of the small and large intestine are not very definite; but if the stools are greyish-yellow and not numerous, and are mixed with partially digested food, and if colicky pains are severe, and if the urine shows a burgundy-red colour, on the addition of nitric acid, and this persists after boiling, indicating the presence of indican, the chances are in favour of the small intestine being the site of the lesion.

Care should always be exercised in deciding whether any given case of enteritis is primary or secondary; that is to say, whether it is the only and primary lesion, or whether it is but a symptom of another disease (*vide* "Infra-chronic and Secondary Enteritis").

Prognosis.—Prognosis is as a rule favourable. The majority of cases recover in a few days after the cause has been removed. In the case of ptomaine-poisoning recovery as a rule occurs, though many cases are serious and demand investigation by the local authorities. Epidemic enteritis of children should always be regarded seriously, for it is remarkable that children apparently but slightly ill in the course of a day or two lose ground so rapidly that they die before one has almost time to realise that any serious danger exists.

Treatment.—The most important matter in the treatment of all cases of intestinal catarrh is to ascertain its cause and so determine whether the remedy should be directed to the removal of an irritant or to subduing the irritability of the inflamed mucous membrane. Slight cases practically cure themselves; the diarrhœa being salutary removes the irritant naturally. After this, restriction in diet or abstention from food temporarily, and perhaps a small dose of opium (pulv. ipecac. co. grs. v. in an adult) may be all that is required. If the cause is an irritant, such as undigested food, a good treatment is to administer a dose of castor oil with 8 to 15 mms. of tr. of opium, and to follow this by a further dose of opium (pulv. ipecac. co. grs. v., and repeated as necessary) to allay peristalsis and give the intestines rest. Having removed the irritant, if the symptoms still persist on account of resulting inflammation, or having determined that the case is one of enteritis not caused by an irritant lying in the bowel, recourse must be had to a combination of sedatives and astringents. Mist. cretæ of the B.P. finds most favour, and may be given alone or in combination with opium, as the mistura cretæ cum opio with kino or catechu added. Chalk acts by saponifying the acid fats, and thus removes acid irritants, at the same time coating the inflamed mucous membrane with a protective and soothing covering in a similar manner to the heavy insoluble bismuth salts (bismuthi trisnit. grs. x., pulv. ipecac. co. grs. v. 4 ter hor.) which are also very useful remedies. If colicky pains are severe, nothing gives such speedy and satisfactory relief as a hypodermic injection of morphia, and with this may be combined the application of warmth in the form of fomentations covered by a waterproof, or a hot poultice over flannel on the abdomen, either to be changed frequently.

If the case under consideration is one of diarrhœa from eating spoilt food or decomposing meat, a dose of castor oil (℥j.) should be administered; but if any of the irritating meal remains in the stomach, it should first be ejected by an emetic. Agonising colic and muscular cramp demand a hypodermic injection of morphia, and a stimulant is required to combat the reflex cardiac failure and the depressing action of the ptomaine on the nervous system. In the case of mushroom-poisoning the antidote to the active principle, muscarine, is belladonna or atropine, and this should be given in 10 or 15 mm. doses of the tinct. belladonnæ, or one-hundredth of a grain of atropine every hour until dryness of the mouth and pupillary dilatation are detected. In enteritis, the result of arsenical poisoning, if there is reason to believe that the arsenic has not already been absorbed, or in other words if the case is seen early, ferric hydrate should at once be freely given; but if the case is seen late, the enteritis resulting can only be treated on general principles. Ferric hydrate can be readily prepared by the addition of liquor ammon. fort. to either the tr. or liquor ferri perchlor., care being taken that excess of ammonia is not present before drinking.

The important part played by micro-organisms in many cases of enteritis has given rise to a useful method of treatment which consists, after the initial administration of a purgative, in the administration of one or other of a class of drugs known as intestinal antiseptics, and of frequent alteration in the diet. By means of the drug we hope to inhibit the growth of the pathogenic bacteria, and by alteration of the diet to starve the bacteria by depriving them of the pabulum on which they feed and multiply. The most useful intestinal antiseptics are the preparations of mercury, in the form of either liquor hydrarg. perchlor., hydrarg. cum cretæ, or calomel. Of these, liquor hydrarg. perchlor. is by many regarded as the best. Calomel, in spite of evidence that it diminishes bacterial putrefaction in the intestine, as shown by the increased amount of bile pigment in the fæces, which has not been converted into urobilin in the large intestine, and by the diminution of aromatic sulphates in the urine, has the drawback that it acts as a purgative partly by increasing the flow of bile, and also by being an irritant, but it is often apparently useful. The salicylic acid group is excellent, and probably salol is preferable to salicylic acid or beta naphthol or salicylate of bismuth. Mercury may be advantageously combined with bismuth and soda, as in the following for an adult:—

℞ Liquor hydrarg. perchlor. ♏ xx.; sodæ bicarb. bismuthi subnit. āā, grs. v.; mucilaginis, q.s.; aqua anethi, ℥ss. Ft. mist.

Or, if salol is preferred, it may be given to an adult in doses of 10 to 15 grains, alone, or with carbonate or salicylate of bismuth. Salol has this great advantage in enteritis, that it passes through the stomach unchanged and breaks up in the intestine into salicylic acid and phenol. Resorcin in doses of 2 to 5 grs. is sometimes used in combination with salicylate of bismuth.

With regard to the second part of this method of treatment, viz. frequent alteration of diet, if the child is bottle-fed, ordinary milk should at once be stopped and recourse had to sterile milk. If this is unsatisfactory, milk should be replaced by animal broths, such as beef or veal extracts prepared at home, or given in the form of Liebig's extract, or similar commercial preparations, with a carbohydrate, *e.g.* ground rice. A very useful form of beef tea is prepared by the cold method. This is prepared by adding

10 drops of dilute hydrochloric acid to half a pound of finely minced beef and half a pint of cold water, which are allowed to stand for an hour and are then strained through muslin. The desired quantity is then placed in a cup and warmed (not boiled) until the colour changes to the brown of ordinary beef tea. These alternations of diet should, in the event of the continuation of symptoms, not last longer than twenty-four hours, when further changes should be made to milk again, milk whey mixed with white of egg and cream, etc.

Chronic or Secondary Catarrh—Etiology.—(1) Acute primary catarrh may become chronic, especially in old and feeble subjects; (2) extension of inflammation from structures adjacent to the intestine; (3) from tuberculous or chronic ulceration of the gut (*vide* "Ulcers of the Intestine"); (4) The result of portal obstruction, hepatic or cardiac; and (5) in pyæmia, etc.

Morbid Anatomy.—The intestinal mucous membrane becomes thickened, fibrous, eroded, and studded with ulcers. The blood-vessels are congested; the muscular coat is generally, to some extent at least, hypertrophied, and later is replaced by fibrous tissue. The glands are destroyed, their ducts being in the first place blocked, and retention cysts form before their final destruction. The villi, as a rule, are shrunken and atrophied.

Symptoms.—The acute form merges into the chronic gradually; the bodily health depreciates; the complexion becomes muddy; there is persistent diarrhœa; appetite is impaired; nausea is present; anæmia and loss of weight follow, and absorption of poisons from the intestinal canal cause listlessness and hypochondria. Constipation alternates with diarrhœa. This form of enteritis is more usually located in the large than in the small intestine. Prognosis is bad, for this is a secondary disease, occurring in the later stages of Bright's disease, heart disease, chronic pyæmia, etc.

Treatment is palliative; and the astringent and sedative remedies used in acute intestinal catarrh are employed. A bland, easily digested food, which yields but little residue, is desirable. Tonics, such as quinine and iron, if there is much anæmia, are useful. Change of scene with hydropathic treatment, are very useful. Each case of chronic enteritis must be treated more on the principle of removal or amelioration of its original causative disease than by the administration of remedies acting on the intestinal tract.

Ulcers of the Intestine

(1) *Tuberculous Ulcers of the Intestine.*—Tuberculous infection of the intestine occurs as a primary or as a secondary disease. As a primary disease it is most frequently found in children, though it occurs in adults, in either case the tubercle bacilli being introduced through the medium of tuberculous milk, etc. As a secondary disease the most common mode of infection is by swallowing of phthisical sputum. It is, however, evident that any medium containing tubercle bacilli when swallowed may be the starting-point of tuberculous ulceration of the bowels. The intestine may also be the site of a miliary tubercle deposited there in a case of general tuberculosis. It by no means follows that an individual who has swallowed one or many doses of tubercle bacilli gets intestinal tubercle. The bacilli must find conditions favourable to its growth, and in an individual predisposed.

Certain pre-existing lesions of the intestine appear to favour the deposition and growth of the tubercle bacillus, *e.g.* follicular ulcers, chronic non-inflammatory catarrh, etc.

Infection may also occur by extension from the peritoneum or by the discharge of tuberculous pus into the bowel.

Situation.—Tuberculous ulceration of the intestine is least rare in the cæcum and lower part of the ileum, and diminishes in frequency above and below this point, the duodenum being but rarely affected. The rarity of tuberculous lesions in the higher part of the intestine is attributed to the destructive or inhibitory effect of the acid of the stomach and upper part of the bowel on the tubercle bacillus.

Anatomical Characteristics.—Tuberculosis arising in the mucosa appears first as a number of little, hard nodular projections in the lymph follicles. The tips of the nodules then become grey and caseate, break down and separate, forming a number of small ragged ulcers. If the caseating nodules lie close together, they coalesce, or if a Peyer's patch is the site an ulcer is formed having the following characteristics:—The floor, formed of the submucosa, is rough, infiltrated, inflamed, and presents caseous nodules. The edges are sinuous and irregular, and slightly undermined. Tuberculous deposit and infiltration extend beyond the edge of the ulcer. The mucous membrane beyond the ulcer is for a certain distance inflamed, but if the ulcers are not close together healthy mucous membrane may intervene. The direction of the ulcer is generally transverse to the long axis of the intestine, and it is in shape oblong, but it may be round. Looked at from the outside of the bowel, the floor of the ulcer is seen to be thickened, and frequently small stellate cicatrices are visible, showing that the serous and muscular coats are involved. Adhesions may occur to the neighbouring coils from spreading inflammation, and perforation seldom occurs. Tuberculous are distinguished from typhoid ulcers by their transverse direction, by their irregular, ragged, and infiltrated edges, by their thickened bases, and by the existence of small caseous nodules in their proximity. Tuberculous disease begins in the lymph follicles, spreads by the lymphatics and infects the mesenteric lymph glands. The ultimate result of tuberculous ulceration is, in the vast majority of cases, death, because it usually occurs in the excavating stages of phthisis. Fifty per cent of phthisical patients have ulcers of the intestine. They may, though rarely, perforate, or may heal and cause stricture of the intestine at one or many points. More rarely healing probably takes place without their existence being suspected, and without leaving noticeable permanent damage to the intestine during life.

Symptoms.— If persistent diarrhœa occurs in phthisis, tuberculous ulceration of the intestine is the likely cause. Tuberculous ulceration, however, does not always produce diarrhœa, and as in non-tuberculous inflammation of the intestine, speaking generally, diarrhœa is more likely to occur the nearer the ulceration is to the rectum. Tuberculous ulceration of the large intestine nearly always gives rise to diarrhœa. Blood may be present in the motions, which are frequently fœtid on account of the bacterial putrefaction, and for the same reason tympanitis is often marked. Tubercle bacilli can, as a rule, be detected in the stools in cases of tuberculous ulceration of the intestine, but their absence is not positive proof of the absence of tuberculous disease. In children, the subjects of primary tuberculous ulceration of the intestine, diarrhœa attended by loss of weight and strength out of proportion to its severity, loss of appetite and sickness, with abdominal tenderness and distension, and the existence of palpable glands in the abdomen, may make a diagnosis possible; but in the absence, after the lapse of some time, of other evidence of tuberculous affection, it is often impossible to have a positive opinion.

Diagnosis.—Lardaceous disease of the intestine may give rise to diarrhœa and recourse for diagnostic purposes may be had to the examination of the fæces for tubercle bacilli, and if these are regularly found in the stools, a diagnosis of tuberculous ulceration is justifiable. As lardaceous disease frequently coexists with phthisis, it is not unlikely that diarrhœa occurring in the last stage may be caused both by tuberculous and lardaceous disease.

Treatment.—The majority of cases occur in connection with pulmonary tuberculosis, and the intestinal lesion is both late and secondary. Remedies to alleviate the diarrhœa are the most generally applicable. The primary disease requires first attention, and in most cases this treatment consists in restraining the diarrhœa, diminishing the fermentation in the intestine, and regulating the diet, so as to avoid as much as possible exciting fresh inflammation in the highly susceptible intestine. For the diarrhœa, astringents are always useful, and of these chalk and opium, kino and catechu, give the best results. Opium by the mouth occasionally causes vomiting, and may then be tried as a suppository or an enema. Salol, in five or ten grain doses, is sometimes useful, as it restrains the diarrhœa and diminishes putrefactive fermentation and flatulence. It is said to inhibit the action of the putrefactive as well as the tubercle bacilli.

Attention to diet as laid down under "Enteritis" is useful, and it is well to give sterile foods, avoiding every article of diet calculated to irritate the intestine. By these means we may hope not only to restrain diarrhœa but to promote the healing of ulcers, and by avoiding fermentation to diminish abdominal distension and the risk of perforation. If much abdominal discomfort exists, warmth, applied to the abdomen as a poultice fomentation, or dry heat, is comforting. If general peritonitis exists, opium should be given freely if the case is hopeless, but it is possible that an occasional case of perforation from a tuberculous ulcer might be treated by abdominal section, with the object of cleansing the peritoneum, and either of stitching the perforation or resecting the bowel at the site of the lesion. In children with primary ulceration of the intestine, the source of infection should be ascertained if possible, and perhaps fresh invasions be avoided by the substitution of sterile food. Diarrhœa is to be restrained by suitable astringents, and means taken to improve the nutrition of the child by general hygienic measures, cod-liver oil, maltine, etc. Strict directions should be given to the mother to ensure the proper ventilation of the nursery, the disinfection of stools, the leading of an out-of-door life in a suitable locality, and, in fact, the case should be regarded as one of grave tuberculous disease, and treated on the general principles now finding so much favour in the modern treatment of consumption.

(2) *Ulcers of the Duodenum* occur as:—

(*a*) Acute primary ulcers; and

(*b*) Chronic secondary ulcers.

(*a*) *Acute primary ulcer* of the duodenum has the same pathology as acute gastric ulcer, and the only points necessary to consider in connection with it are the modification of symptoms and sequelæ in virtue of its different site.

(*b*) Chronic secondary ulcer of the duodenum occurs most frequently after severe burns, and these, indeed, constitute the most important group of this class, but they also, though rarely, occur in portal congestion, specific fevers, and in septicæmia. It is not understood why ulcers occur in the duodenum after severe burns, nor is the rationale of their production explained. As in gastric ulcer, peptic digestion is rendered possible by the lowered nutrition of a limited area of the mucous membrane, and several theories have been advanced to account for this. Septic emboli in the duodenal vessels are said to occur, but have never been demonstrated microscopically, and artificial emboli experimentally introduced into the circulation rarely affect the stomach and duodenum, but when affected they are equally so, whereas ulcers following burns occur more frequently in the duodenum than in the stomach. Hunter has shown that when toluylidiamine is subcutaneously injected in the dog, it is eliminated by the bile, and causes duodenitis and ulceration of the duodenal mucous membrane. He therefore suggests that toxic irritating products absorbed from burns are eliminated by the bile and cause similar lesions. Against this theory is the fact quoted by Fenwick that similar lesions occur if the bile-duct is ligatured, and also that after burns ulcers occasionally occur in the stomach. Burns, it is known, are accompanied by inflammation of the mucous membrane of the bowels, and also by gastritis. This may be sufficient explanation of duodenal ulceration, but Fenwick suggests that the stomach and intestines may eliminate poisonous septic products which act as irritants, in the same manner as urea injected subcutaneously appears in the stomach and is found as crystals. The same authority quotes an experiment in which he injected albuminose prepared from septic sloughs of burns, and by these means produced "appreciable duodenitis."

Situation.—The most usual site of duodenal ulcer is between the opening of the bile and pancreatic duct and the pylorus, on the anterior and mesial side of the duodenum. These ulcers diminish in frequency near the bile duct, probably because peptic digestion ceases after contact with bile or pancreatic juice.

Symptoms.—For convenience of arrangement the symptoms of both classes will be first considered, and their distinctive characters subsequently. Duodenal ulcers generally produce obscure symptoms. Pain varying in character from uneasiness to violent and prolonged suffering occurs in more than half the cases. It occurs two to five hours after food, is situated well to the right of the middle line of the abdomen in the right hypochondrium, and is frequently relieved by sitting up with the body bent forward. The explanation of its occurrence two to five hours after food is that the contents of the stomach reach the duodenum at this period and cause pain by irritation of the ulcer.

Vomiting is much less than in gastric ulceration, and bears no relation to the severity of the pain. Hæmorrhage from the ulcer may be observed either as hæmatemesis or melæna. Fenwick found that hæmatemesis occurred alone or with melæna in an equal number of cases, and that melæna occurred in seven out of seventeen cases where there was evidence that the ulcer had bled. Perforation followed by peritonitis occurs in more than half the cases (53 per cent), and is accompanied by the same symptoms as perforation by a gastric ulcer, and death by general peritonitis is a frequent result. The peritonitis has, however, a better chance of becoming localised in duodenal than in gastric ulcer, for the escaping contents are poured first into the liver pouch described by Rutherford Morison, and may become localised there by adhesions. Perforation of ulcers of the duodenum may also result in a peri-duodenal abscess, or the ulcer may perforate into a neighbouring coil of bowel.

Of the differential symptoms, the following are noteworthy :—

Acute primary ulcer of the duodenum, which is a rare disease, occurs in contradistinction to gastric ulcer much more frequently in adult men than in young women. It usually gives rise to no marked symptoms until sudden melæna, hæmatemesis, or perforation declares its presence. It should be noted in this connection that if hæmorrhage occurs in duodenal ulcer, it is likely to be profuse, because the blood-vessels eroded are usually large trunks (pancreatico-duodenal or gastro-duodenal), and that perforation is very liable to take place on account of the thinness of the duodenal wall. Perforation followed by peritonitis is said to occur in 90 per cent of the cases. Fenwick says it occurred in all his cases.

Secondary ulcers following burns commence a few days after the burns, develop slowly, and reach their maximum in the second week. It is not uncommon to find a small quantity of blood in the dejecta of cases of burning, but this does not necessarily imply ulceration. The enteritis which accompanies many cases is in itself sufficient to explain this. Melæna and hæmatemesis occur in 30 per cent of the fatal cases, and bleeding, which generally occurs from the pancreatico-duodenal artery, is often fatal. Perforation is rare because the ulcer is indurated, and adhesions to the neighbouring tissues have had time to form. The tendency of these ulcers is to cicatrise, and a scar with subsequent stenosis or a pouch of the duodenum may result.

Diagnosis.—Diagnosis is frequently difficult and often impossible. Duodenal ulcers may be distinguished from gastric ulcers by the following points:—

(*a*) Duodenal ulcer occurs much more frequently in men than in women, and rather later in life.

(*b*) Pain is less severe and occurs two to five hours after food; it is referred to the right hypochondriac region, and seldom to the back.

(*c*) Hæmorrhage is very frequent, and melæna with hæmatemesis is most suggestive of duodenal ulcer.

(*d*) Vomiting is independent of food and does not relieve the pain.

(*e*) Perforation is extremely common in acute primary ulcers.

The symptoms of duodenal ulcer may simulate gall-stone colic, but the occurrence of melæna and a hæmatemesis would serve as distinguishing features, and if jaundice was absent in hepatic colic, the gall-bladder should form a palpable tumour.

Treatment.—Except in the chronic forms of ulceration from burns, where hæmorrhage has occurred, drugs are useless, but in this instance some reliance may be placed on half-drachm doses of turpentine given in a gelatine capsule or in milk. The administration of ergot by the mouth, or the hypodermic injection of ergotine, might also be tried. If the symptoms are not urgent and point to cicatrisation of the ulcer, strict rest in bed, the blandest of food, or the substitution of rectal feeding, should be enjoined. If there is reason to suppose that perforation has taken place, or if hæmorrhage has occurred more than once and is exhausting the patient's strength, prompt abdominal section offers the best prospect, as by this means there is a fair chance that the perforation may be discovered and general peritonitis prevented, or the bleeding vessels may be secured.

(3) *Syphilitic Ulceration.*—Syphilitic ulceration rarely occurs in the intestine. Ulceration and stricture, condylomata and mucous patches are well known in the rectum, and various pharyngeal lesions constitute well-marked features of syphilis, but the proximal and distal ends of the digestive tract are almost exclusively affected. In infantile syphilis ulcerative plaques and small gummata have been found, but their presence as a clinical feature is not established, a remark equally applicable to acquired syphilis. In severe and neglected cases of syphilis, with gummata elsewhere in the body, diarrhœa occurs, and may be due to broken-down

gummatous ulcers in the intestine. In such circumstances before arriving at a diagnosis of ulcer the question of lardaceous disease of the intestine, and the possibility of tuberculous ulceration in a syphilitic subject, must be excluded. The treatment of syphilitic ulceration of the intestine is that of the general disease.

(4) *Catarrhal or Follicular Ulcers.*—These are found in inflamed conditions of sufficient severity of the intestinal mucous membrane, and are more common in the large than in the small intestine. They occur either as simple erosions of the mucous membrane apart from the intestinal glands, or as the result of inflammation and destruction of the solitary, Lieberkühn's, or Brunner's glands. Both varieties are usually present, it may be in large numbers, in the more severe cases of enteritis. Their presence is usually indicated by blood in the stools. Small ulcers are also found above the site of intestinal obstruction, but the pathology of them is still doubtful. The treatment of follicular and catarrhal ulcers is that of enteritis.

(5) *Malignant Ulcers* (*vide* under "Malignant Diseases of the Intestine," p. 433).

(6) *Hæmorrhagic Ulcers.*—In Bright's disease hæmorrhage into various organs is liable to occur, and in the intestine the chosen site is under the mucous membrane. This submucous hæmorrhage instead of being absorbed may break down and form an intestinal ulcer.

Ulcers of the intestines in Bright's disease are usually not single but multiple. As enteritis is not an uncommon complication of kidney disease, it is therefore questionable whether the intestinal ulcers of Bright's disease should be regarded as broken-down submucous hæmorrhages or as catarrhal ulcers occurring in an enteritis complicating the original disease. Both varieties probably occur, and it is also probable that in purpura, scurvy, anæmia, etc., intestinal ulcers of purely hæmorrhagic origin develop.

(7) *Vascular ulcers* are either venous or arterial. Whether venous or arterial they depend on defective blood-supply to an area more or less limited, this leading to necrosis of the intestinal mucous membrane and the subsequent formation of an ulcer.

Venous ulcers occur in cirrhosis of the liver where the portal circulation is impeded, and probably have the same pathology as ulcers of the leg connected with defective venous circulation.

Ulcers due to arterial blocking are described. The artery may be occluded in either of the three following ways:—

(*a*) Plugging of the mesenteric vessel or one of its branches by emboli. This variety may complicate a case of endocarditis. The symptoms would probably be intense abdominal pain and tenderness coming on suddenly, followed, if the patient lives long enough, by bloody stools, tympanites, and peritonitis.

(*b*) The mesenteric artery, or one of its branches, may be the site of thrombosis; or

(*c*) Atheroma may obstruct the lumen, and circulation becomes so defective that numerous ulcers of the mucous membrane result. The pressure of a tumour on a vessel may effect the same result.

Lardaceous Disease.—The intestine is affected by lardaceous disease next in frequency to the kidneys, spleen, and liver. If it does occur at all, it is extremely rare for the intestine to be the only structure involved; indeed, they do not usually become waxy until the disease is far advanced in the solid organs. The intestine in 30 to 40 per cent of cases of lardaceous disease is affected.

Morbid Anatomy.—As in other organs the amyloid changes commence in the small arteries. In mild cases the change is confined to the mucous and submucous coats, the villi being specially affected, but all coats may become involved. To the naked eye the mucous membrane presents a glossy translucency, is pale and somewhat swollen, but the iodine test is usually required to settle the question, when the characteristic reaction will be found as spots and lines distributed irregularly over the surface. If the waxy disease is advanced, the mucous membrane may break down and form small ulcers. The small intestine, especially the lower end of the ileum, is the most usual seat of the disease.

Symptoms.—The most prominent symptom is diarrhœa with or without hæmorrhage. The occurrence of a persistent diarrhœa is usually the first indication that the intestine has become waxy, and the occurrence of this symptom in a case where other organs are known to be waxy is strong evidence of this. If the stomach is also waxy vomiting may occur, but the possibility of the vomiting being uræmic should always be remembered. Abdominal pain and tenderness are rare.

Diagnosis.—It is always difficult and often impossible to distinguish lardaceous disease of the intestine from tubercular in a given case where either or both may occur, as in the later stages of phthisis. It is only by consideration of each case in all its bearings that an opinion can be formed.

Treatment.—If possible the causative disease should be removed, *e.g.* syphilis, tubercle suppurating joints, etc. In this the only hope of cure lies. In the great majority of cases all we can hope to do is to restrain the diarrhœa by astringents, etc. (*vide* also "Lardaceous Disease," vol. vi.).

MALIGNANT DISEASE OF THE INTESTINE.—Malignant growths of the intestines differ but slightly from malignant disease of other parts, in so far as the characteristics of the growths are concerned, but when a hollow tube is attacked the potency of every part of which is recognised as important, and when the dependence of health on a vigorous digestive apparatus is remembered, the special importance of this subject will be admitted.

Site.—Of 1148 cases collected by Ewald—

874 occurred in the rectum.
148 occurred in the large intestine (of these twelve occurred in the transverse colon).
64 in the cæcum, including the appendix.
26 in the ileum.
19 in the duodenum.
17 in the jejunum.

In the above Table, cancer of the small intestine (duodenum, jejunum and ileum), as compared with the large intestine (cæcum, appendix, colon, sigmoid flexure and rectum), occurs in the proportion of 1 to 17½. It will thus be seen that the large intestine is much more frequently affected than the small, and that the jejunum is the portion least likely to be involved. Statistics of other authors show the jejunum to be more rarely involved than the above. It is a noteworthy fact that where the intestinal contents stagnate longest (rectum), or pass through a part with diminished calibre, or where two differing surfaces adjoin (ileo-cæcal valve and pylorus), there cancer is most frequently found.

Pathological Anatomy.—Carcinoma of the intestine may be primary or secondary. Primary is far more common than secondary cancer, which, indeed, is rare, and occurs either by metastasis, or by extension from neighbouring organs (pancreas, uterus, stomach, etc.), and being but a complication of pre-existing disease, requires no special mention here. Primary cancer of the intestine occurs as cylindrical-celled epithelioma, more rarely as a spheroidal-celled growth. Either of these forms may undergo colloid degeneration and produce a third variety known as colloid cancer.

Cancer of the intestine begins as a small deposit in the mucous membrane, usually in Lieberkühn's glands. As it increases in size it spreads in such a manner as to present itself in three distinct aspects, namely:—

1. As a nodule, which, expanding equally in all directions, becomes a more or less rounded tumour, the intestinal surface of which is apt, from irritation of the intestinal contents and bacterial invasion, to become ulcerated and assume a polypoid outline.

2. The growth may spread laterally rather than towards the lumen of the bowel, and assume the form of a plaque or raised flattened mass on one side only of the intestinal wall, with well-marked edges, and often ulcerated centre.

3. Most commonly extension takes place in an annular manner, so that the intestine is surrounded by a ring of cancerous deposit often of small longitudinal depth. This variety is the commonest cause of malignant stricture. It occurs most frequently in the large intestine, and is a frequent cause of intestinal obstruction. In the later stages ulceration at the site of growth, dilatation of the bowel above the stricture, ulceration at or above the stricture, and probably hæmorrhage, mark the subsequent course. Extension early takes place to lymphatic glands, and secondary deposits occur most usually in the liver, and other neighbouring abdominal organs, the peritoneum becoming invaded by direct extension; or ulceration and perforation may occur into the bladder, stomach, and other parts of the intestine, or into the peritoneum, generally or becomes localised by adhesions. In some cases of annular stricture gangrene of the cancerous deposit is apt to occur. The author has seen a case of annular stricture of the sigmoid which caused intestinal obstruction, necessitating colotomy. The case ended fatally by peritonitis, due to gangrene of the annular stricture with subsequent perforation. The stricture was so insignificant in size that it was impossible to say definitely without microscopic investigation, and the corroborative and extensive secondary deposit in the liver, that it was a malignant one.

A few special points are noteworthy in cancer of the duodenum, which is usually columnar-celled, but may be spheroidal-celled. The growth begins in the mucous membrane, or possibly in Brunner's glands, though the latter origin is doubtful. A malignant growth of the head of the pancreas and of the lower end of the bile and pancreatic ducts or the biliary ampulla is an extra-duodenal growth, in which the duodenal mucous membrane is not, until the last stage, and may not be at all, involved. Any part of the duodenum may be the site of primary cancerous growth, but it is important to distinguish from a clinical point of view whether the original site is above or below the biliary papilla. Malignant growths of the duodenum, as elsewhere in the intestine, have a tendency to produce annular stricture resulting in dilatation of the duodenum and stomach behind the stricture, and the symptoms simulate those of pyloric stricture.

Symptoms.—The presence of a malignant growth in the intestine may be revealed in a variety of ways, but a diagnosis in the early stages is rarely possible. The initial subjective and objective symptoms seldom occur until the growth has existed for some time. It may happen that acute intestinal obstruction will be the first sign to arouse suspicion, a condition of affairs explicable, and caused by the sudden blocking of the intestinal lumen at the site of growth by hard scybala, or foreign bodies. Perforation of the bowel with peritonitis may be the first intimation of serious mischief. The symptoms, however, generally develop slowly. Vague dyspeptic symptoms, a sense of discomfort in the abdomen, even amounting to pain, constipation alternating with diarrhœa, a distaste for food, mental irritability, nausea, vomiting and progressive loss of strength, with cancerous cachexia, usually precede the more definite symptoms of intestinal blocking. The assertion that if a patient over thirty-five years of age complains of persistent dyspeptic symptoms for the first time, cancer of the stomach should be suspected, is equally applicable to cancer of the intestine, with perhaps this addition: that the sufferer from intestinal cancer will probably also complain of flatulence and diarrhœa alternating with constipation. Unfortunately, the early symptoms of cancer, until it has attained to the stage of a palpable tumour, are always vague and uncertain. If a tumour of the small intestine can be detected, it may occupy any part of the abdominal cavity, because, with the exception of the second and third part of the duodenum, and the colon, the intestine is attached to a longer or shorter mesentery, which allows of considerable displacement by the weight of the tumour itself, or by pressure communicated to it from without. The tumour may vary in size, or be more readily felt at one time than another, due to the amount of abdominal distension or presence of fæcal masses behind a stricture. The stools frequently contain blood, and, indeed, considerable hæmorrhage may take place from the ulcerated mucous membrane. For further symptoms of intestinal obstruction in Malignant Disease, *vide* p. 434.

In cancer of duodenum, the general symptoms irrespective of the part affected are wasting, the gradual appearance of cancerous cachexia, anorexia, nausea, and vomiting at variable intervals after meals, pain in the right hypochondrium, hæmatemesis, rarely melæna, and in the later stages signs of dilatation of the stomach from stenosis of the duodenum, will usually be present. In the later stages, after ulceration of the mucous membrane has allowed of auto-intoxication from bacterial absorption, the temperature rises. If the growth invades the duodenum above the biliary papilla, which usually enters the duodenum three or four inches below the pylorus, the vomited matter is unlikely to contain bile, and the symptoms are similar to those found in cancer of the pylorus. If the growth is below the biliary papilla, bile and pancreatic juice regurgitate into the stomach, and their presence in the vomit can be easily recognised. Bile may also in this case be deficient in the stools. In supra-papillary cancer the tumour is movable, and resembles a pyloric cancer, whereas in the infra-papillary variety it is fixed, and may be mistaken for cancer of the head of the pancreas. The growth may occur at or so near to the biliary papilla itself as to produce symptoms indistinguishable from those brought about by cancer of the common duct, or its ampulla, where jaundice is an early symptom.

Diagnosis.—Diagnosis is never easy, and in the early stages, in the absence of a palpable tumour without an exploratory laparotomy, it is impossible. If an abdominal tumour is absent, the persistence of symptoms in spite of treatment, the age of the patient, progressive loss of weight, and the appearance of cancerous cachexia, are the chief symptoms to rely on, but it is often necessary in the early stages to arrive at an opinion by a process of exclusion. After the appearance of a tumour great care has still to be exercised before a definite opinion can be formed.

Cancer of the first part of the duodenum is practically indistinguishable from cancer of the pylorus, but differs from simple ulcer of the stomach by the presence of a tumour, by the small quantity of blood vomited, and by pain occurring some hours after food.

Malignant disease of the duodenum below the pyloric papilla is distinguished from cancer of the head of the pancreas or of the lower end of the bile-duct by the absence of jaundice and by the presence of signs of duodenal stenosis and gastric dilatation.

If the case is one affecting the intestine below the duodenum, we must first ascertain by the administration of enemata and purgatives whether, after all fæcal accumulation is removed, any tumour is left. In every case rectal examination is then made, and in women, if possible, vaginal also. The history of the case and careful weighing of the symptoms may aid in the differential diagnosis from other conditions producing abdominal tumours, especially malignant disease of the uterine appendages, hydronephrosis, floating kidney, and tuberculosis of the peritoneum, and from other causes of intestinal obstruction, such as healed typhoid and tuberculous ulcers, and the impaction of gall-stones.

Treatment.—Medical treatment can only be palliative. Surgical operation should receive the first consideration, because a radical cure of malignant intestinal growths is now no longer impossible. The diet should be so regulated that no irritating matter is allowed and little fæcal residue results. It should be chiefly fluid, easy of digestion and absorption. By this means the chance of intestinal obstruction is diminished, pain is lessened, a greater degree of rest is secured, and the tendency to hæmorrhage from the ulcerated mucous membrane is obviated. In advanced cases it may be advisable to rely largely on rectal feeding. In the last stages of the disease, when all hope of recovery has passed and the patient's life is rendered miserable by constant suffering, opium or morphia should be regularly given in sufficient doses to secure relief and peace.

In cancer of the duodenum with symptoms of stenosis and dilatation of the stomach, lavage gives temporary relief, and gastro-enterostomy will permanently relieve the distressing vomiting and prolong life. Intestinal antiseptics, such as salol, appear to do good, if fever and other symptoms of auto-intoxication are present. For hæmorrhage, turpentine or ergot may be tried.

In cancer of the small or large intestine, excision of a limited growth offers the chance of a radical cure, and is in any case the best palliative. Excision of growths of the large intestine has given the best results, and exploratory operations are advisable as soon as there is a reasonable doubt of their presence. If the disease has advanced too far to admit of excision of the growth, an intestinal anastomosis performed at the situation most suitable to the particular case will allow of a temporary return to health and remove the obstructive symptoms (*vide* "Intestinal Obstruction," p. 426).

Sarcoma of the Intestine.—Sarcoma of the intestine is more rare than carcinoma. Few cases have been reported, and it is difficult to estimate exactly how many of the reported cases are true sarcomata, sarcoma, lymphosarcoma, and lymphoma being confused in the descriptions. Lymphosarcoma occurs in the intestine in connection with Hodgkin's disease, and may grow to a large size. Sarcoma here, as elsewhere, may occur at an early age, and cases have been reported in children of a few years of age. The infiltration of the bowel rarely produces stenosis or obstruction; on the contrary, the lumen is usually made larger by the growth. Constitutional symptoms and wasting appear early, and death occurs after a shorter illness than in carcinoma. The prognosis and the results of treatment are equally bad.

NON-MALIGNANT TUMOURS OF THE INTESTINE.—*Adenomata.*—Adenomata arise from Lieberkühn's or Brunner's glands, and have a typical acinous structure. A considerable amount of fibrous tissue may be found in their matrix when they are fibrous, or they may be myxomatous and soft. They may be attached to the mucous membrane by a broad base or by a narrow pedicle of variable length. The large intestine is their most frequent site, and considerable numbers may be present.

Fibromata.—Fibromata arise from the connective tissue of the submucous coat, are always numerous, usually small, and may be either sessile or pedunculated.

Myomata and Fibromyomata arise from the muscular coat, are usually small, and possess a pedicle.

Lipomata and Angeiomata also occur, but are rare.

All the above are usually classified as intestinal polypi. Clinically these innocent tumours may give rise to no symptoms, or to irregular attacks of diarrhœa, accompanied by hæmorrhage.

Their weight may cause invagination or intussusception, and if large, they may produce mechanical obstruction. Cases have been recorded where they have become detached and passed per rectum, making the diagnosis clear.

ACTINOMYCOSIS OF THE INTESTINES is a rare disease. One mode of infection, *i.e.* of the deposition and growth of the essential fungus, is through the intestinal mucous membrane. The lesions may occur in any part of the intestine, but recorded cases point to the large intestine as the most usual site, the cæcum and appendix being probably the most frequent. The early lesions consist of small abscesses in the submucous coat, apparent to the naked eye only as indefinite semi-translucent swellings, which, when punctured, contain pus and the small spherules of the fungus. These abscess cavities either rupture into the intestine and form an ulcer, which may heal and leave a puckered cicatrix, or, more usually, infiltrate and destroy the muscular coat, acting as foci of a spreading chronic inflammatory induration, which extends to the peritoneum and causes matting of the surrounding parts, interspersed with small abscess cavities. A palpable tumour is thus formed, which may, and usually does, run a chronic course, tends to involve the abdominal wall and to open externally, but when it does so, fails to discharge pus in quantity like an ordinary abscess. In the discharge the characteristic granules are found. The process is essentially a chronic spreading inflammation with suppuration, which may, by virtue of its situation by extension or metastasis, spread to the liver, kidneys, etc., or may by a fistulous opening communicate with the bladder, stomach, or other part of the intestine.

Symptoms are always obscure, and many cases remain doubtful or unsuspected until a necropsy reveals the true nature of the disease. In the early stages the symptoms are usually those of intestinal irritation and atony—diarrhœa of variable severity and abdominal distension from flatulence. As the disease advances a palpable tumour becomes evident. According to the situation this will resemble an appendix abscess, a sarcoma, etc., and unless it is possible to find granules in the stools, urine, or discharge of the abscess, it is exceedingly difficult to form an opinion as to the nature of the case. Temperature is rarely elevated, and the course of the disease is strikingly slow unless some complication such as a perforation and peritonitis should supervene.

Diagnosis.—Careful search must always be made for the characteristic spherules of the fungus in the stools, in the discharge of abscess if the case has arrived at this stage, and in the urine. On the detection of these only can a positive opinion be formed. Ransom says—"When a subacute or chronic inflammation occurs which spreads without much elective affinity for any tissue or viscus, which is hard, even woody, to the touch, involves the skin, softens in the centre slowly, at length yields pus in scanty proportions relative to the bulk of the swelling, and does not heal when freely opened and drained, it is almost certainly an infective malady, and actinomycosis may be strongly suspected, and the spherules and colonies should be sought for."

Treatment.—Large doses of iodide of potassium, at least one drachm daily, is the most hopeful remedy, and, it is said, may cure. Ransom reports such a case. Intestinal antiseptics also afford relief. If a palpable tumour exist it should be freely opened and drained. If circumstances permit, it is possible that scraping or any means to eradicate the then apparent lesion are justifiable, in spite of the fact that other organs or tissues may be involved. (*Vide* also "Actinomycosis" and "Appendicitis.")

LITERATURE.—**General**: ALLBUTT'S *System of Medicine*, "Diseases of the Intestine." **Special**: MUIR and RITCHIE'S *Text-Book of Bacteriology*; CRUIKSHANK'S *Text-Book of Bacteriology*; S. and W. S. FENWICK, *Ulcers of the Duodenum and Stomach*. **Actinomycosis**: RANSOME, *Med.-Chir. Trans.* vol. lxxv.; LEITH, *Edin. Hosp. Reports*, vol. ii.

Intestines, Surgical Affections of.

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INTESTINAL obstruction is caused only by mechanical obstacles, and surgical consideration is demanded from the outset. A sudden complete obstruction of any part of the small or large intestine causes the same serious symptoms as are met with in strangulated hernia, and the pathological conditions produced above the obstruction (and these are far the most important) are the same in both. It is therefore obvious that if as early surgical treatment could be adopted in cases of intestinal obstruction as in cases of strangulated hernia, the present bad results, which every surgeon deplores, would at once improve. The difficulty of early diagnosis, more than anything else, contributes to fatal delay. A careful and intelligent practitioner can scarcely make any mistake when he sees and feels a strangulated hernia, but the diagnosis of a similar condition hidden in the abdomen is surrounded by difficulties.

For the first few hours in a case of sudden complete obstruction, the symptoms produced are those brought about by any condition which may cause a profound impression on the abdominal nerves, and are those of abdominal shock. Many serious abdominal lesions, such as perforated gastric or duodenal ulcer, ruptured vermiform appendix, leaking ectopic gestation, gall-stones, etc., are attended by symptoms which are indistinguishable from those occurring in the early stage of sudden obstruction. That they are due to nervous influence in cases of obstruction is proved by experiments on animals, which show that vomiting and other acute manifestations do not follow artificial constriction of the intestine when the mesenteric nerves supplying it have been previously divided. This knowledge also helps to an understanding of the marvellous relief following the use of opium.

The second stage soon follows on the first. Shock is recovered from, and the typical symptoms and signs of obstruction are now more likely to be met with than at any other time, if carefully looked for. These are produced by a vigorous attempt on the part of the intestine to get rid of the obstruction by forcible contractions in its unstriated muscular coat. The forcible contraction of unstriated muscle, here as elsewhere, induces the most agonising pain, in great part intermittent, and many patients already more or less familiar with intestinal pain, as most of us are, will at this stage express a confident belief that if only wind could be voided they would soon be well again. Shortly, and not slowly, the vigorous contractions cease; the intestines become more distended, gas and fluid increasing, and the third stage of intestinal paralysis is reached. This is accompanied by venous engorgement in the intestine above the obstruction, most marked in its immediate neighbourhood, but rapidly involving higher and higher portions, due to interference with the blood circulation by distension. Following the circulatory disturbance there is an increased quantity of fluid poured into the intestine, absorption being at the same time checked. Increased gas formation from the putrid contents helps to further the distension. Free fluid is also found in the peritoneal cavity, and in the most acute cases may contain sufficient blood to colour it, as occurs in the sac of an acute strangulated hernia. The intestinal wall above the obstruction becomes red, thickened, sodden, and friable, and the first histological alteration apparent in it is an active destruction of the cells lining its mucous membrane, which play so large a part in its protection from septic invasion. Below the obstruction the bowel is pale and contracted. The bacterium coli now becomes exceptionally active, thriving in the stagnating intestinal fluids and invading the intestinal walls. The transition from this stage to the final one, if the patient survives the auto-intoxication produced by absorption from his own intestines, and exhaustion resulting from the starvation, pain, and vomiting, is by peritonitis, brought about either by the migration of organisms through the distended and damaged intestinal walls, or by the more gross method of perforation through a distension ulcer or gangrenous patch above the obstruction. In the final stage the only diagnosis possible may be that of peritonitis.

CLASSIFICATION

No classification is entirely satisfactory. Perhaps the best is one which divides the causes of mechanical obstruction into five classes:—

1. Strangulation of the intestine by bands, accidental rings, internal herniæ, adhesions.

2. Defects of position in parts of the intestine, such as intussusception, volvulus, kinks, displacement by tumours, etc.
3. Blocking of the lumen by foreign bodies, polypi, hard fæcal masses, etc.
4. Strictures, simple and malignant.
5. Paralytic ileus.

1. *Strangulation by Bands, Internal Herniæ, Adhesions, etc.*—Strangulation by bands appears to have become more common since abdominal section ceased to be a rare operation, for many of these bands result from a preceding peritonitis, and may be caused by the traumatisn of operation. In addition to this cause, disease in the appendix or in the pelvic viscera, or tuberculous lesions in the abdomen, are frequent sources of a peritonitis which leaves adhesions to be stretched and thinned into cords or broader bands. Another origin is not uncommon: a tag of omentum adhering in the neighbourhood of a femoral or inguinal hernia in course of time, by a process of stretching and rolling, is converted into a cord or band, often of a great length. Such cords or bands produced by any of the causes mentioned may strangle the intestine in every conceivable way, so much so that no two cases are exactly alike. Adhering at both ends, either to viscera or parietes, and remaining free in the centre, a bridge is formed under which a loop of intestine may slip and be strangled. A long cord may loop itself and become knotted round an intestinal loop, as a piece of string purposely tied round it would. On the other hand, the intestine may wind itself round such a cord, and become strangled by its own act. More than one case has been reported where two bands have been found post mortem, the wrong one having been divided during operation. In addition to the band, it is not infrequent for a volvulus to be found in the ensnared intestine beyond the band. Many of the well-marked cords are not, however, due to a preceding peritonitis, but have a congenital origin from persistent remnants of the omphalo-mesenteric vessels. In this connection, too, the Meckel's diverticulum may be noticed. In the human fœtus, about the eighth week of intra-uterine life, the vitelline canal normally disappears. If it does not, the connection between the small intestine, usually at the convex surface of the ileum, and not far from the ileo-cæcal valve, and the umbilicus, may permanently remain. Then a tubular off-shoot or diverticulum possessed of the same structures as go to form the small intestine may be found extending from the intestine to the umbilicus. More commonly a portion of this tube is obliterated, and there is found projecting from the intestine a short diverticulum attached by a cord to the neighbourhood of the umbilicus. At times the distal extremity of the diverticulum, or its obliterated representative, is set free and may continue to float, or may contract fresh adhesions to any part of the parietes or viscera. Another band producing precisely similar results may be constituted by the adhesion of normal structures, for example the vermiform appendix, the Fallopian tube, the appendices epiploicæ, the intestine itself, and its mesentery. Cases also are recorded of intestine strangulated by the long pedicle of an ovarian cyst.

The pathological results produced in the intestine by strangulating bands are of two kinds:—(1) Those limited to the area of intestine strangulated; and (2) changes in the intestine above. The latter are similar in every instance of acute obstruction, and have already been referred to. The local changes are those produced directly by the constricting agent, causing necrosis from pressure at the site of constriction, and vascular changes in the gut beyond, which tend to produce gangrene of the portion ensnared. An early release of the gut shows a pallid depression at the point constricted, the pallor and constriction disappearing in a few minutes after its release. At a later stage the pallid constriction does not disappear, and section of the intestine shows that the whole thickness of its wall, except the peritoneal coat, has been destroyed, the site of the constriction internally being marked by ulceration of the mucous and muscular coats. This ulceration is found in the entering portion of gut, and at the limit of the distended portion when the constriction is a small tight band, and only a few inches of gut have passed under it, as is generally the case (Fig. 1); but not uncommonly it occurs at the further limit of the ensnared gut, as when a large coil of intestine passes under a loose band, and is obstructed only at its outlet (Fig. 2). Evidences of local peritonitis are soon seen in the strangulated loop. Its surface becomes dull and sticky, and later on is covered with purulent lymph. Finally, the signs of gangrene appear. The lowest portion of the ileum is most commonly caught in this form of obstruction.

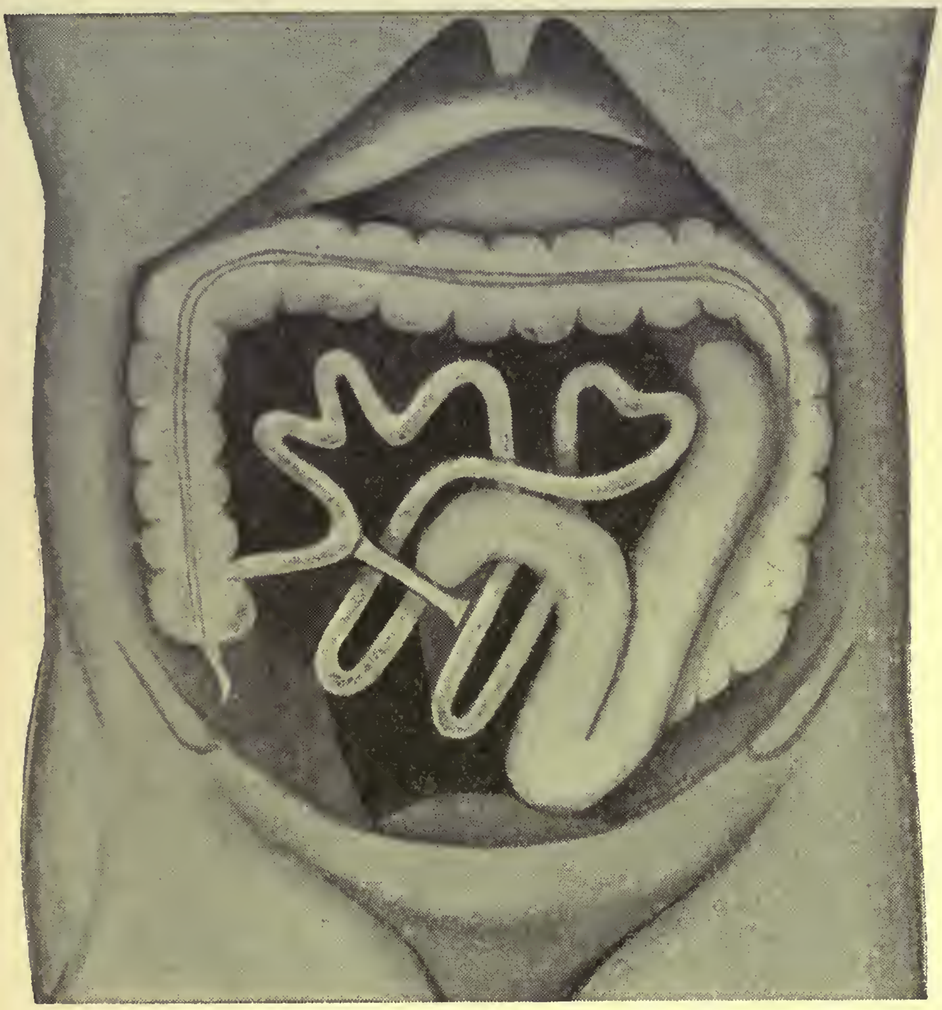

Fig. 1.—Strangulation by tight band.

Internal Herniæ.—In the peritoneum, covering the posterior abdominal wall, chiefly at the duodeno-jejunal and ileo-cæcal junctions, and in relation to the cæcum and sigmoid flexure, pouches of peritoneum are normally found. Into one of these a portion of bowel may enter. Indeed, cases are recorded in which the whole of the small intestine has been found in such a sac. The gut may be held and become strangulated there; or a loop of intestine may pass through either normal or abnormal openings in the mesentery or omentum, such as are occasionally found in healthy subjects, or such as may be left after tears from injury, or the foramen of Winslow may allow the gut to pass through and become retained in the lesser peritoneal sac, strangling it at its entrance; or as the result of a congenital or accidental opening, intestines may pass into the pleura through the diaphragm and be nipped by the congenital or acquired orifice. Diaphragmatic hernia is practically limited to the left side, as congenital openings are generally found there, and the liver protects the right side from the effects of injury.

Adhesions.—Adhesions occur often as a result of preceding peritonitis, due chiefly to tuberculosis, appendicitis, and, in women, to pelvic lesions. In the latter case the results are somewhat different from those produced by the ordinary bands or adhesions. A loop of intestine becomes adherent to some portion of the pelvic wall, frequently the bladder or uterus. Under the bridge of intestine and mesentery so formed, coils of intestine find their way between the arch of intestine and mesentery in front and the pelvic wall behind. The intestinal contents pass freely into the ensnared loops, but have difficulty in making their escape. Distension follows, and the increased tension brought about by this forces the exit channel into a position from which it cannot escape, usually the sharp angle formed by the mesentery, and this portion of intestine becomes acutely strangulated as by a band (see Fig. 2). Kinking of the intestine may cause obstruction by the dragging of a limited adhesion (Fig. 3), producing a valvular occlusion, or so much matting may attend extensive adhesions that the passage of intestinal contents is prevented.

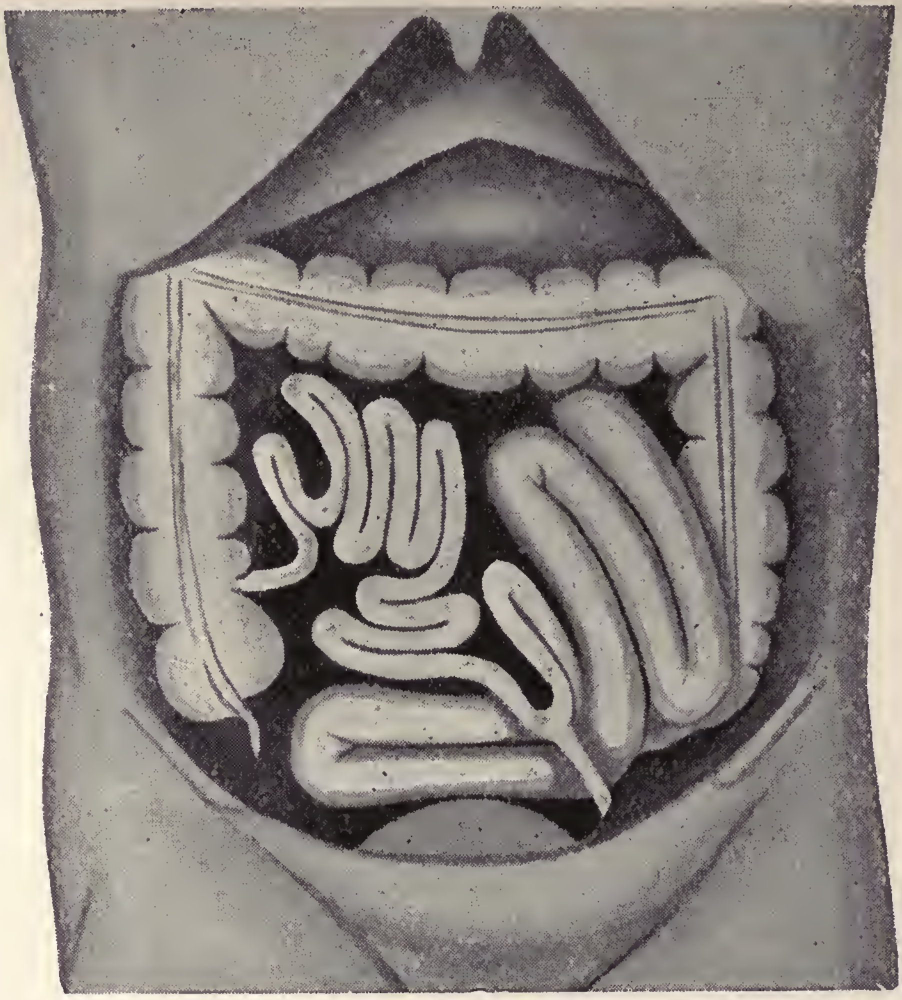

Fig. 2.—Strangulation by loose band.

2. *Defects in Position of Parts of the Intestines.*—Intussusception is the most common example of this variety, and one of the frequent causes (about one-third of all cases) of intestinal obstruction. A portion of constricted bowel is received into the cylinder below it, so that a transverse section through the involved portion from without inwards would show (1) the sheath with its mucous surface in contact with the mucous surface of (2) the returning or middle layer, the peritoneal coat of which is in contact with the peritoneal coat of (3) the entering layer. The inner two layers are

known as the intussusceptum, the outer or sheath as the intussuscipiens. The upper part is known as the neck or collar, and the apex is the lower extremity of the intussusceptum. The mesentery is found between the layers of the intussusceptum, and assists in the arrest of further progress of the involved portion of intestine by the traction it exerts. In addition to this the intussusceptum is made to take a curved form by it, and the orifice at its apex to become more slit-like than circular. Any part of the small or large intestine may be involved, and these varieties are described under the name of the portion of intestine affected.

Intussusception in children before they are five years of age is common. It appears to follow frequently the vigorous, irregular peristalsis caused by purgative medicine or irritating foods, and is seldom due to any organic disease. In adults it is comparatively rare, and with few exceptions is occasioned by growths in the bowel, either polypi or annular malignant growths. The ileo-cæcal valve certainly plays a very important part in its production, as the ileo-cæcal junction is the commonest site of it. This valve has been aptly compared to the anus, and intussusception here to prolapse of the rectum. I have seen acute intussusception follow immediately on severe strain in a healthy young adult, and in babies, too, vigorous jumping up and down previous to the attack has been observed in so many cases that traumatism must be noted as an exciting cause.

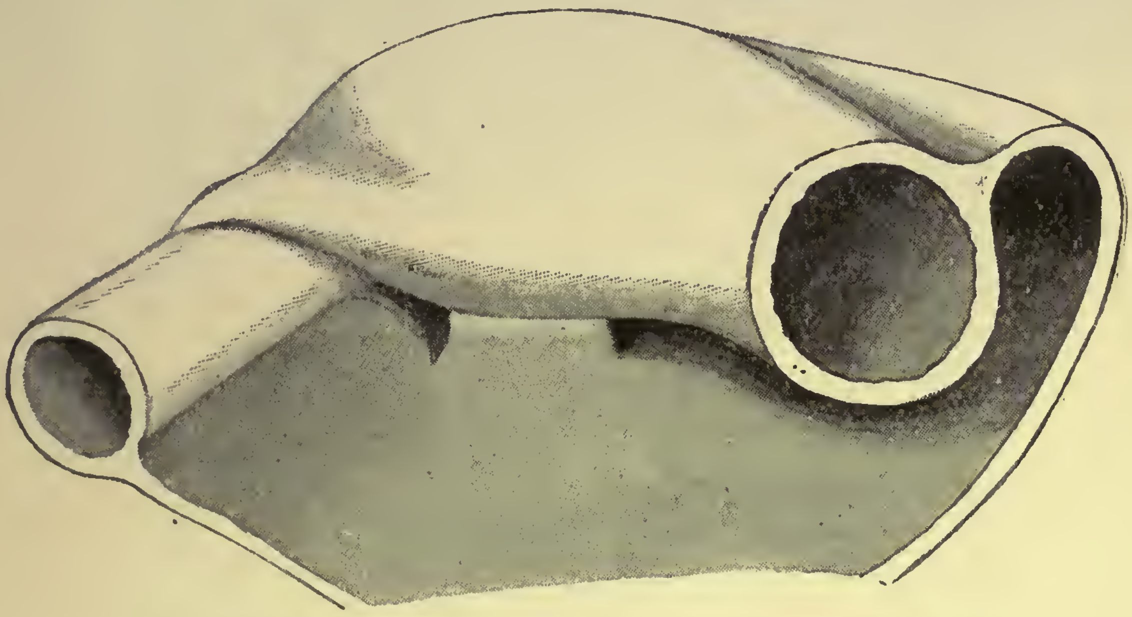

FIG. 3.—Kink with adhesion to a tuberculous mesenteric gland.

Apart from the intussusceptions found post-mortem, which produce no obstruction and cause no symptoms, and appear in the act of dying, two distinct varieties of intussusception, pathological and clinical, are met with: the acute and the chronic. In acute cases the intussuscepted bowel and its mesentery are nipped by the collar at the neck, and the intussusceptum is affected as is the imprisoned bowel in a strangulated hernia: First, The venous circulation is obstructed in the intussusceptum; then follows œdema and consequent interference with the arterial supply; next the damaged tissues yield to the attack of the bacillus coli, and become inflamed, gangrene occurring as the final result. Above the intussusception the intestine is distended, and destructive circulatory changes are brought about in it as in all obstruction cases. The most important pathological facts to bear in mind from the clinical point of view are the swelling of the intussusceptum produced by venous engorgement, which in the course of a few hours may be so pronounced as to make reduction impossible. The engorgement is most marked at the apex of the intussusceptum, which becomes so swollen, rounded, and firm as to resemble a tumour, and to effectually resist ordinary efforts made for its release. Of even greater importance is the fact that adhesions are likely to form rapidly in acute cases, and to glue together the peritoneal coats of the involved intestine. These adhesions, though favourable to spontaneous recovery by sloughing of the intussusceptum, are a hindrance to its surgical reduction. The usual causes of death are gangrene of the sheath, perforation in it or in the bowel above, and gangrene of the intussusceptum.

In the chronic variety there is at first little if any interference with the vascular supply of the involved bowel, and inflammation is consequently not brought about. Even after many months in some chronic cases reduction has been easily effected, on account of there being but a small amount of swelling and an absence of inflammatory adhesions to glue the displaced and opposed peritoneal surfaces together. Sooner or later, however, in these cases more active mischief develops. Frequently, in the neighbourhood of the lesion, ulcers form, and perforation with peritonitis terminates existence. The gut above is frequently hypertrophied as in other chronic obstruction cases.

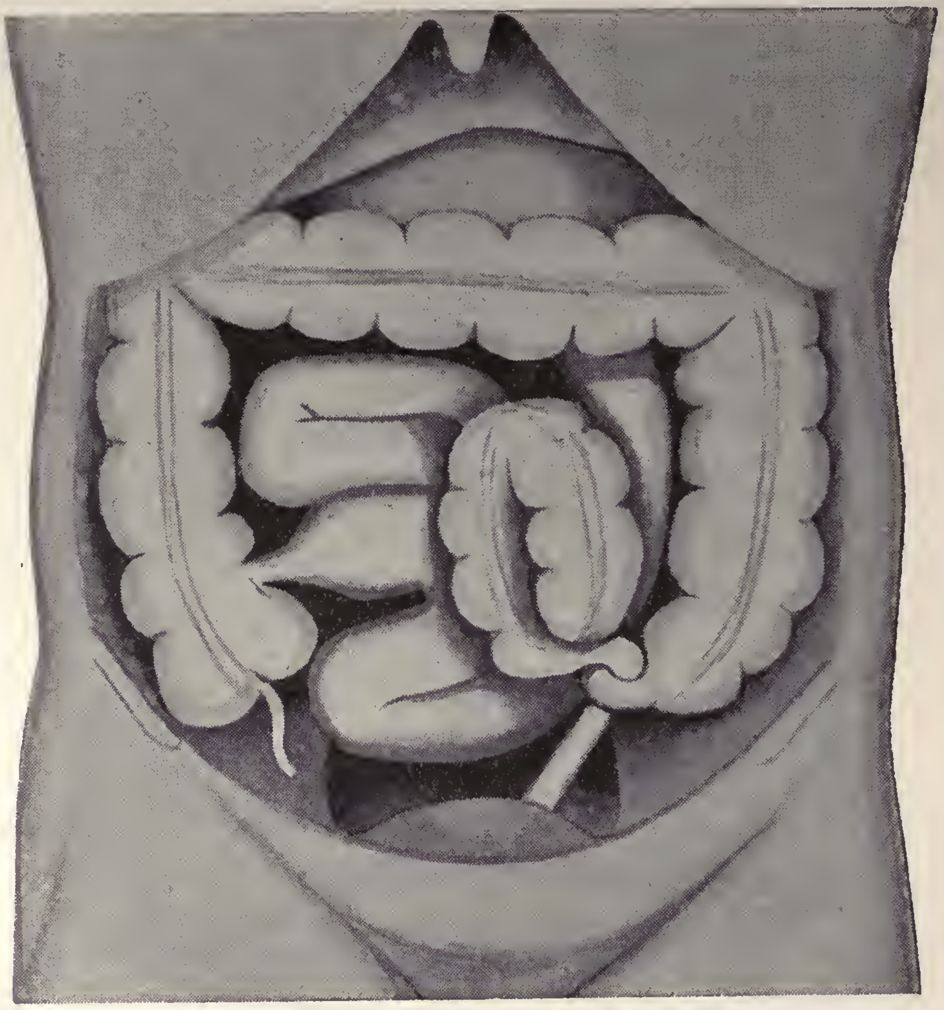

FIG. 4.—Volvulus of sigmoid flexure.

Torsions and Volvulus.—Torsions and volvulus, though frequently spoken of as common, are quite rare. With a sufficiently long mesentery the most complicated knots and twists may arise, but except for the puzzling difficulties in dealing with these complicated conditions surgically, they are of small clinical importance.

Volvulus is a well-marked variety. It consists in the twisting of an intestinal loop round an axis formed by its mesentery, a congenital or acquired lengthening of which is commonly found associated with it (Fig. 4). It chiefly occurs in elderly adults, and the sigmoid flexure is its most common site. For this there are several reasons: the sigmoid is a long loop with its commencement and termination close together. The loop has consequently a narrow neck, and in patients suffering from habitual constipation is filled with heavy matter, which tends to elongate its mesentery.

Volvulus is one of the most acute varieties of intestinal obstruction. By torsion of the mesentery the circulation of blood in the involved intestinal loop is suddenly interrupted. The intestine becomes of a plum colour, and rapidly distends with gas and fluids. Obstruction is caused by the pressure produced by one end of the loop pressing upon the other end. The distension of the sigmoid loop may become so great that the whole abdomen appears to be occupied by it, and the diaphragm may be pushed up, pressing upon and displacing the heart. Organisms soon find their way through the damaged and softened gut, and peritonitis as the result of this transmigration frequently causes death. If the patient lives long enough small patches of gangrene appear in the gut, and by perforation of these rapidly fatal peritonitis may be caused; but this, owing to the speedy fatal issue, is a rare termination. I have recently met with a rare case in which volvulus of the sigmoid was the cause of chronic obstruction.

Kinks.—Kinks are often the result of fixation of an intestinal loop by adhesion, and occur at the junction of the fixed with the movable portion (see Fig. 3). At other times they result from the union of two loops, as when a strangulated hernia is reduced and the damaged bowel is retained by inflammatory adhesions in the position held by it in the sac.

The possibility of displacement and compression of the intestine by tumours external to it, though rare, ought to be borne in mind. Tumours of the ovary, of the uterus, of the broad ligament, of the pancreas, of the liver, of the spleen, of the kidney, and inflammatory masses, have all been noted as causes of intestinal obstruction.

3. *Blocking of the Lumen from within.*—The most common cause of this accident is a large *gall-stone,* which usually escapes into the small intestine by a direct fistulous communication between the gall-bladder and the duodenum. It would appear unlikely that a gall-stone small enough to get through the common bile duct could obstruct the intestine. The stone may be arrested at any part of its course through the small intestine, but its usual place of arrest is in the lower ileum. If it passes into the large intestine the dangers are as a rule over, though I have operated on a case of intestinal obstruction produced by a gall-stone blocking and causing inflammation of the sigmoid flexure, and have had to remove from the rectum a large gall-stone which could not pass the anal sphincter, and was causing serious obstruction symptoms. Hard fæcal masses, clumps of cherry-stones, intestinal calculi, and a variety of foreign bodies, introduced either by the mouth or rectum, are also reported as causes of obstruction.

Polypi may be benign or malignant, and single or multiple. Next to the rectum they are most frequently seen in the colon or ileum. Obstruction may be caused directly by the tumour or tumours blocking the intestine, or by their producing intussusception through dragging on their attachments.

4. *Strictures.*—Strictures are simple and malignant, the latter chiefly predominating.

Malignant strictures are most commonly found at and after middle life, and in the large intestine. In the small intestine they are comparatively rare. Not uncommon sites are the ileo-cæcal valve, the hepatic and splenic flexures of the colon, and the sigmoid flexure. Apart from the rectum, they occur with the greatest frequency in the sigmoid flexure (in 60 per cent of cases). The stricture is brought about by an annular growth, sometimes remarkably limited, of cylindrical-celled carcinoma, which contracts the bowel more and more as it grows. Cancerous growths of the bowel wall and growing into its lumen occasionally obstruct it, though not causing genuine stricture, and the same is true of other growths, such as lipomata, fibromyomata, polypi, adenomata, sarcomata, and cysts, all of which, though rare, have been found.

Simple strictures are commonly found in the small intestine, and are comparatively rare in the large. They occur mostly in young people, and their commonest cause is a healed tuberculous ulcer in the neighbourhood of the ileo-cæcal valve. Other causes are: congenital defects, usually in the portion of ileum occupied by Meckel's diverticulum, when they are probably due to an excess of the developmental involution by which the process is normally obliterated, traumatism, contractions subsequent to ulceration of a portion of strangulated gut, and contractions following syphilitic ulceration, which selects by preference the large intestine. Whether the stricture be simple or malignant, the form of obstruction caused by it is usually chronic, and then dilatation with hypertrophy of the walls of the intestine above the stricture is produced by it, and is an important clinical feature. The cæcum, especially in these cases, may reach such a size as to extend far into the left side. The mucous membrane of the strictured colon is frequently in a catarrhal state owing to the injury produced by putrefaction and mechanical irritation by the fæcal contents of the bowel. This offers an explanation of the frequency with which patients suffering from stricture complain of diarrhœa, which may be the most noticeable symptom. Pouching of the intestine above the stricture is not uncommon, and foreign bodies are frequently found in these pouches.

5. *Paralytic Ileus.*—After abdominal section or the apparently successful reduction of a strangulated hernia, or a severe painful accident, such as fracture of the femur, due to indirect violence, the patient may die in from two to ten days with distended painful abdomen, vomiting, in the end fæcal, and complete or partial intestinal obstruction. I have made careful post-mortem examinations on several of these cases, and beyond extreme distension of the intestines have discovered nothing. Such a condition is known as "Paralytic ileus," and the name expresses probably all that is known as regards its pathology. After every abdominal section there is more or less intestinal distension, accompanied by irregular peristaltic contractions causing severe griping pains, and it is rare for a patient to be able unaided during the first two or three days to pass flatus or motion. If this amount of distension, which may be regarded as normal, should increase, the position of the intestines in the abdomen becomes more and more cramped, till in the final stages active mechanical obstruction is caused every few feet by acute bends in the intestinal tube. Hence in the end the difficulty is due in great part to a mechanical cause.

The Diagnosis

Cases of intestinal obstruction are divided clinically into two classes, the acute and the chronic; but classical text-book varieties will comparatively rarely be seen. The diagnosis is based upon certain symptoms, and in some cases, unfortunately not in all, on certain definite physical signs.

The Symptoms.—Acute obstruction makes its appearance suddenly. Pain occurs as the first symptom, and is very severe, sufficiently so in many instances to cause marked shock. The pain is accompanied by sickness, which is shortly followed by vomiting. The vomited matter consists, at first, of the contents of the stomach and clear fluid, then bile-stained fluid, and finally the vomit becomes fæcal in character. The depression is more marked the more acute the attack. Ordinary symptoms of shock are present; the patient is pale and chilly, has a feeble pulse and drawn face, and a faint voice. There is complete constipation; no wind can be passed, and hiccough may be a troublesome symptom. The abdomen in this stage is retracted, and kept fixed in the endeavour to relieve the pain. The most lasting symptoms are distressing thirst and vomiting.

Pain.—Pain may be local at first, and it is possible that the patient may be able by its help to point out the neighbourhood of the obstruction; later, as a rule, the pain becomes more ill-defined, and is apt to be referred to the neighbourhood of the umbilicus. Although the pain may never be absent, there are, as a rule, intermittent paroxysms during which it is markedly increased. The pain is not aggravated, but may be relieved by pressure.

Local Tenderness.—Tenderness is of greater importance than pain as a localising symptom. It is frequently to be found over the site of the obstruction, and occasionally the contracting gut may be felt to harden at this spot.

Vomiting.—Vomiting is frequent in the early stages of the illness, but in the later becomes less so, though it continues throughout, and in acute cases is one of the most important and troublesome symptoms. It depends to some extent on the nervous organisation of the patient, occurring more frequently in children and in women than in strong-willed men. Vomited matter, with fæcal odour, is the most characteristic sign of intestinal obstruction. The large quantity brought up, the obstinacy with which the vomiting persists, and the force with which the vomited matter is ejected, are also strongly suggestive signs. Fæcal vomiting is so rarely met with in other conditions that special importance is always attached to it. It occurs, however, in septic peritonitis, but even then it is probably due to mechanical obstruction caused by distension and kinking of the intestines. In two cases of malignant disease involving the stomach and transverse colon, and causing a fistulous communication between them, which have come under my observation, it was present.

The tongue is usually moist and foul in the early stages, and there is fearful thirst; later, the tongue becomes dry.

Constipation.—Constipation at first may not be complete, because it is possible for the bowel below the seat of the obstruction to empty itself of matter contained in it, though this is very rare. Just before death a motion is not infrequently passed. Rarely, though it is important to bear the fact in mind, diarrhœa may be the most prominent symptom of obstruction. The most marked case of this sort that I have seen occurred in a woman on whom I operated for stricture of the ileo-cæcal valve. Every two weeks she had an attack of diarrhœa, attended by severe griping pains, with marked gurgling and peristaltic contractions and bad-smelling vomit. The lower ileum was enormously hypertrophied and dilated, and at the time of operation (an interval), though every effort had been made to clear her intestinal tract, with apparent success, by purgatives, the small intestine, though not distended, contained several pints of fæcal matter. It appeared to me probable that this patient's ileum was never able to empty itself thoroughly, and that the attacks of diarrhœa every fortnight were explained by the discharge of a large quantity of irritating matter, gradually accumulated there, into the colon. Spurious diarrhœa, with tenesmus, is a not uncommon symptom of stricture far down the colon.

Passage of Flatus.—Flatus is never passed except in cases of intussusception or partial obstruction, even though the bowel be relieved of solid matter. In intussusception, along with blood and mucus, a small quantity of flatus may pass during the attack of tenesmus.

Temperature.—Temperature is normal or subnormal, and this may be an important diagnostic point.

Swelling of the Abdomen.—Though in the early stage the abdomen is contracted to relieve pain, a little later swelling occurs, and this may increase rapidly and impede respiration. In cases of partial and chronic obstruction the hypertrophied and distended intestine may be seen and felt contracting and dilating through the abdominal wall. This sign is pathognomonic of intestinal obstruction. It may also be possible to locate the neighbourhood of the obstruction if it can be ascertained that the contraction ends always at some definite spot.

A tumour may be found in intussusception and in fæcal obstruction; also in obstruction due to foreign bodies, cancer, tubercle, matting of adhesions, internal hernia, tumours pressing on the gut; and sometimes the empty coils of intestine pressed down into the pelvis, can be felt from the rectum as an indefinite swelling.

Chronic obstruction begins gradually. It is often preceded by a period of loss of health and weight, and dyspepsia with constipation. The constipation is at first entirely relieved by a purge, but a characteristic history is one which tells of increasing rumblings of wind and pain after the administration of a purge. Later the patient may emit wind, though no stool passes. In cancer there is sometimes blood, spurious diarrhœa, and tenesmus. In chronic intussusception the same symptoms may be present. The stools, when the large intestine is diseased, may be altered in shape, and become tape-like or small worm-like in form. Frequently, on exposing the abdomen, contractions of the dilated and hypertrophied colon are visible. Vomiting occurs in the later stages, but fæcal vomiting is generally long delayed. The pain is intermittent, and is dependent upon forcible contractions of the intestine above the obstruction. The general symptoms are wanting at first. The end of the case is usually brought about by an acute attack supervening on the chronic course of the disease.

In a variety of acute abdominal lesions the symptoms are similar; for example, in peritonitis due to perforation of any of the viscera, in ovarian tumour with twisted pedicle, in ruptured or leaking ectopic gestation, in an attack of gall-stones or renal colic, in cholera, and in certain kidney cases; so that the first question to be answered on seeing such cases is:—

1. *Is it one of intestinal obstruction?*

The history is of value. If the attack be due to appendicitis there is frequently a history of previous ones, and it is rare for the perforative variety of appendicitis to start quite abruptly. If due to the perforation of a gastric ulcer there will probably be a history of previous stomach troubles. In ruptured or leaking ectopic gestation there may be the usual history of pregnancy, a missed menstrual period; if to an ovarian tumour, the tumour may have been observed before its pedicle became twisted. Perforating typhoid ulcer is preceded by illness of a febrile character. In kidney cases there may be albumin in, or total suppression of the urine. If the attack be due to gall-stones, a history pointing to attacks of hepatic colic, followed by jaundice, may be elicited.

The diagnosis from peritonitis is especially difficult. In peritonitis the pain is more rapidly generalised and more superficial; there is more abdominal tenderness; the abdomen swells more rapidly; the muscles become markedly rigid; pain and tenderness are increased by pressure, and the vomiting is less abundant and more rarely fæcal than in obstruction; the constipation is also less complete. In peritonitis the temperature is high at first, and later it may be lower than normal. In obstruction it is lower at first, and may be higher later when peritonitis develops. In peritonitis there may be signs of peritoneal effusion, and the abdomen becomes dull in all the most dependent parts. In obstruction intestinal movements may be visible.

The diagnosis, even if the greatest care be taken, is not always possible during the earlier stages.

The case is likely to be one of intestinal obstruction if the patient, previously in good health, has been seized with sudden pain, which is much increased at times, if there is entire inability to pass flatus, and if vomiting is an urgent symptom.

The next proceeding is to make a complete examination. The abdomen should be freely exposed, the patient lying with shoulders raised in an easy posture and in as good a light as possible. The hernial sites are to be first carefully examined, not only the ordinary ones, but the extraordinary as well. An absent testicle on one side would suggest a strangulated hernia, possibly hidden in the inguinal canal.

It is impossible to overrate the importance of a careful examination for hernia in the first place, for the most skilful practitioner, if he should forget this, will sooner or later commit an error fatal to his patient; indeed, I go further, and teach students that the first thing to be done in every case of urgent vomiting is to examine carefully the hernial sites, whether the patient considers it necessary or otherwise, and to believe if a hard lump is found there that this has *everything* to do with the condition until the contrary is proved by operation. The whole abdomen should then be systematically and carefully inspected, palpated, and percussed. A rectal examination must never, under any circumstances, be omitted, and in women, when possible, a vaginal examination should also be made.

2. *Where is the block?*

In acute obstruction the probabilities are that the block is in the small intestine, and in chronic obstruction that the block is in the large. The reason for this is that chronic obstruction is mainly due to stricture of the intestine, and the majority of strictures are malignant and in the large intestine. Though this general rule holds good in the majority of instances it has many exceptions. I have seen acute sudden obstruction produced by malignant strictures at the ileo-cæcal valve, at the hepatic and splenic flexures of the colon, and in the sigmoid flexure; and in one of the most chronic cases that has come under my notice the obstruction was in the middle of the jejunum, and due to adhesion and kinking of the bowel.

Pain localised by the patient may be of use as a guide to the site of the obstruction if the localisation is made quite early in the case. Pain, however, is a doubtful help, as it is mostly referred to the neighbourhood of the umbilicus. A tender spot is a much more certain guide, and becomes of great importance if a swelling or feeling of resistance is felt in its neighbourhood. In chronic obstruction the patient may feel pain which begins or ends at one spot, and flatus rumbling in his intestines may be felt by him to stop there. On inspecting the abdomen, in exceptional cases, moving intestinal coils may be seen, and their movements may be directed to one particular spot; and on palpation the peristaltic wave may be felt to be arrested there, producing a feeling of temporary firm tumour under the examining hand. This, in chronic cases, is a valuable guide as to the site of the obstruction.

A careful examination should be made in the early stage, because shortly the general swelling of the intestines which takes place is likely to obliterate any landmarks.

The shape of the abdomen is an aid in forming an opinion as to whether the obstruction is situated in the large or in the small intestines. When the small intestine is involved low down the abdomen becomes globular in form and prominent in the middle line, a similar distension to that caused by an ovarian tumour. When the large intestine is involved the abdomen is flattened and bulging at the sides, as in ascites, and may be enormously distended. A flattening at the left side, with enlargement on the right, points to an obstruction in the commencement of the transverse colon, the swelling on the right side being constituted by the distended cæcum and ascending colon.

If it can be made clear that the cæcum is distended the obstruction is certainly below this point. In some cases the distension of the cæcum is quite evident. It may be seen to swell up intermittently, to contract, and to harden, and peristaltic waves spread from it along the ascending colon towards the right costal margin. The abdominal swelling will also, in the early stages, be limited to the right half of the abdomen. There may be splashing on concussion of the cæcum. The pain and contraction are felt by the patient, and may be seen to begin in it.

A ladder-like arrangement of the distended intestine, seen in some chronic cases, is an aid to localisation, for this occurs only when the small intestine is involved. Very firm and painful contractions, in which the distended intestine becomes as hard as the contracting uterus, occur, so far as I have seen, only in the small intestine, for the colon and the stomach contract more slowly and more feebly than the small intestine.

Vomiting in the case of obstruction of the small intestine is more rebellious than in the large, and sooner becomes fæcal in character. In the case of the large intestine vomiting is usually a late symptom, and is rarely fæcal.

The general condition of the patient is more serious in small intestine cases. Depression and feebleness are more marked.

In cases of chronic obstruction of the small intestine anuria is a symptom of value, and is due to limited absorption of fluids and to their loss by vomiting.

Muscular cramps in the feet and legs are mostly met with in acute cases where the small intestine is affected.

The use of an enema I have frequently found to be a great help. If a small one only can be retained (say, less than one quart), this sign is in favour of the obstruction being in the large intestine.

An examination of the patient under chloroform may discover a tumour in one or other part of the abdomen. Distended intestinal coils felt per rectum point to obstruction in the small intestine.

As aids to diagnosis the passage of the long tube or a sound into the rectum are useless, and the introduction of a hand into the rectum is likely to convey very little information, and is a dangerous operation.

3. *What is the nature of the obstruction?*

If the case is one of acute obstruction it is probably due to acute intussusception or volvulus, to strangulation by a band or a ring, or to an internal hernia or gall-stone.

Intussusception.—Intussusception is the simplest form for diagnosis. It occurs most commonly in early youth. The attack is often preceded by a free intestinal evacuation. There is sudden pain—continuous at first, becoming, later, more intermittent. There is vomiting, which is not so severe or so persistent as in other acute cases; constipation, which is not likely to be complete, as it is usual to have small loose motions with muco-sanguinolent discharge and marked rectal tenesmus. The abdomen is seldom much swollen; indeed, it may be retracted, as relief is obtained by the passage of small quantities of gas per anum. Examination of the abdomen may discover at the ileo-cæcal junction, or in the course of the colon, a rounded sausage-shaped tumour, which hardens at times, or there may be merely marked local tenderness. Per rectum, it may be possible to feel the end of the intussusceptum, like a uterine cervix, or, if the tumour is too high for this, bimanual examination with one hand on the abdomen and a finger in the rectum may make it plain. A blood-stain on the examining finger may also give a clue. The swelling in the ileo-cæcal region may be mistaken for the swelling due to an abscess in connection with the appendix; but the history of the onset, the absence of intestinal symptoms characteristic of intussusception, the febrile course, and the tender, indefinite lump, as compared with the rounded and more readily palpable swelling of intussusception, serve as distinguishing features. In young children the diagnosis is occasionally very difficult. The typical symptoms are masked by what I can only describe as apathy. Except for vomiting there are no definite abdominal symptoms. The patients lie in any position in which they may be placed; with sunken features, looking very ill and apparently anæsthetised; they tolerate examination abdominally and rectally without complaint. In consequence of the relaxed and anæsthetic condition of the abdominal wall, the tumour is readily discovered.

Volvulus.—Volvulus occurs in elderly adults with a history of long-standing constipation. The pain and tenderness are most marked in the left iliac region, and there a vague swelling may be felt. Vomiting is not so frequent as in other forms of acute obstruction, probably because older persons are not so frequently sick as young ones are. There is absolute constipation, and frequently rectal tenesmus, but no tumour can be felt on rectal examination, and there is no bloody or mucous discharge. Abdominal distension appears early and rapidly increases. Later it is frequently most marked in the epigastrium.

Strangulation by bands, diverticula, rings, and internal hernia are often sudden in their onset, and produce very acute symptoms. A history of previous peritonitis suggests strangulation by a band or adhesions. In vigorous, previously healthy, young adults, Meckel's diverticulum should be remembered. In subjects of Glénard's disease with gastroptosis, movable kidneys, etc., retro-peritoneal hernia may be suspected. This may form a palpable tumour on the left side of the abdomen. Patients with strangulated diaphragmatic hernia are frequently unable to lie on the left side in consequence of difficulty of breathing, and complain of trouble in their chest. The upper part of the abdomen in these cases may look hollow, and percussion and auscultation yield signs of stomach and intestine in the thorax. In gall-stone cases there is sometimes a previous history of hepatic colic or of peritonitis in the neighbourhood of the liver. Stout old women are the favourite subjects for it. In them the obstruction symptoms may commence acutely, then subside into those of incomplete obstruction, with passage of more or less flatus and partial relief. Not infrequently strictures, malignant or simple, first draw attention to themselves by producing an acute attack of intestinal obstruction, though it is usual on close inquiry to get a history of previous trouble.

In the *chronic form of intestinal obstruction* tumours external to the bowel should be first excluded as the cause. Uterine and ovarian tumours are perhaps the most common. Then chronic peritonitis must be eliminated, and this is difficult when the general abdominal swelling is great. The diagnosis is mostly to be made between the following causes of obstruction:—Fæcal masses, strictures, adhesions, chronic intussusception (for rectal cases see "Rectum").

Fæcal masses are mostly found in feeble old persons, hysterical women, and lunatics. The history is one of long-standing constipation and of gradually increasing abdominal swelling; there are borborygmi and attacks of colicky pain. Vomiting does not occur until late. A series of masses, which are not tender, and which are hard and movable, may be felt in the course of the colon. There is frequently fœtid diarrhœa from the irritation of the mucous membrane of the intestine by hard putrid collections. Tenesmus is marked when the hard lumps pass into the rectum. Rectal examination may reveal scybala there, and help the diagnosis. Purgatives and enemata may not for some time remove the swellings and relieve the symptoms. This form of obstruction is unfortunately regarded as common, and it must be borne in mind that it is easier to mistake malignant masses for hard fæces than the reverse.

Cancer and stricture are usually confounded because the form which cancer takes is frequently annular, and the intestine is in reality strictured by it. In a patient over forty years of age cancer should be suspected. A tumour at the ileo-cæcal valve, or the hepatic or splenic flexures, or in the sigmoid, may be discovered. In women a vaginal examination should seldom be omitted. The sigmoid flexure with a malignant growth in it, from its weight will sink into the lower part of the pelvis, and may be felt there from the vagina, lying at the bottom of the pouch of Douglas. Attention to this point has more than once enabled me to make a definite diagnosis of malignant stricture of the sigmoid flexure. The tumour, if rounded, hard, and nodular, as it frequently is, closely resembles an enlarged and prolapsed ovary. In men a similar tumour of the sigmoid may fall into the lower part of the recto-vesical pouch, and can be recognised by a finger in the rectum, most easily when the patient is standing upright. As a rule constipation is the first symptom. It yields to purgatives in the early stages, but gradually becomes more obstinate, when it may last for some days, and be finally relieved by a purgative and enemata. A very suggestive history is that purgatives caused so much intestinal commotion as to produce rumbling and fluid-dropping sounds loud enough to be heard by any one in close proximity to the patient.

Pain in all chronic cases is characterised by its severe paroxysmal character, and is attended by visible intestinal movements along with the rumblings. After the pain has gone the patient may feel well for weeks or months, but as time goes on the attacks become more frequent and more severe. Towards the end it is usual to have sickness and vomiting. In malignant cases sometimes there is blood found in the stools which are also loose and fœtid. If this occurs late in the history of the case it means ulceration of the growth, and possibly is attended by greater freedom in the passage of the intestinal contents following this destructive process.

In simple stricture a history leading to the diagnosis of tuberculosis, syphilis, dysentery, or typhoid may be obtained, or that the symptoms were preceded by a violent contusion of the abdomen, or by a strangulated hernia. There is a history of attacks of pain usually occurring after food, and frequently attended by vomiting. Associated with these are rumblings of wind and attacks of constipation, or alternately diarrhœa and constipation. The paroxysms become more frequent, vomiting and constipation more severe, and abdominal distension with vigorous peristaltic movements develop.

If a tumour is felt at the ileo-cæcal valve it is difficult to say whether it is tuberculous or cancerous, or due to chronic intussusception. A tuberculous tumour, ill-defined and caky, generally occurs in young subjects with the pallor and weakness that are suggestive of the constitutional feebleness common in tuberculous subjects. It is accompanied or preceded by diarrhœa, and there is frequently a nocturnal rise of temperature, with night sweats. Other evidence of tuberculous taint may be available—the scars of old suppurating neck glands or changes in one or other lung, or the scars of some tuberculous skin, bone, or joint affection.

The cancerous tumour, hard and nodular at the ileo-cæcal valve, occurs in elderly subjects, and is preceded and accompanied by constipation, because of the obstruction which the growth located at the valve causes to the passage of intestinal contents. Occasionally blood will be passed per rectum. The patient may have the cachectic appearance associated in the minds of so many with cancer, but it is quite as common to find the look of robust health.

In chronic intussusception there is intermittent pain; during the intervals the patient may be quite easy. There are muco-sanguinolent motions and tenesmus, and a cylindrical tumour softer than the cancerous or tuberculous one, and perhaps hard at one time, soft at another, and altering in position. Vomiting is not a marked symptom. The bowels are regular, or there may be diarrhœa. There is no general swelling of the abdomen. The patient gradually gets thinner and weaker, and this it is which frequently suggests that the disease is cancer.

Strictures both simple and malignant may become occasionally blocked by hard fæces or foreign bodies, and the patient then develops acute symptoms.

Chronic obstruction, due to adhesions, is suggested by a previous history of peritonitis, possibly tuberculous, or consequent upon pelvic disease, or following an appendicitis. In such cases the symptoms are apt in the early stages to be badly characterised; they develop slowly, and run a course which shows the difficulty of drawing the artificial distinction which we have attempted to do between acute and chronic obstruction. At either end the distinctions are clear enough, but in the great majority of cases of intestinal obstruction it is impossible to say that the acute will remain so till the end, or that the chronic will escape an acute termination, and in these cases the commencement may be acute, and after a day or two of serious illness the symptoms become more chronic, or partially relieved by the passage of flatus.

A distended abdomen with paroxysmal pain and fœtid diarrhœa is seldom met with except in chronic obstruction of the colon, and diarrhœa is indeed so common in cancer of the colon that it is imperative to examine the rectum in every case of obstinate diarrhœa in the adult.

PROGNOSIS

The difficulties of correct diagnosis and, consequent thereon, of exact prognosis have increased the uncertainties of treatment. Before abdominal surgery reached its present position, if there were a fair chance for the case to recover without operation, there was every excuse to delay operative treatment, which had so high a mortality in intestinal obstruction cases. This excuse no longer exists, and it is fair to say now that the mortality after operation is more due to delay, or to diseased conditions which could not have been relieved at an earlier stage, than to the operation itself.

Acute Intestinal Obstruction.—In acute cases the mortality is very high. Strangulation by bands or rings or internal herniæ will have as high a mortality as cases of strangulated hernia would if treated without operation. It is possible for either to get well when apparently in the most desperate condition, but such cases are so rare that they cannot be allowed to count in making the prognosis.

Post-mortem examination made by me of a stout and apparently healthy woman, who died suddenly twelve hours after the onset of obstruction symptoms, showed that death was due to a small knuckle of the ileum being strangulated through a hole in the mesentery. On another occasion in a vigorous young blacksmith, who lived for three weeks after the onset of acute obstruction, death was found to be caused by a band crossing and constricting the end of his ileum.

The great majority of cases die during the first week, and seldom live for more than three days after fæcal vomiting has commenced. Death in these cases is frequently brought about by changes in the intestine above the obstruction. Interference with the circulation, due to distension, first destroys the lining epithelium of the mucous membrane, and allows of the absorption of septic matter into the circulation. Sudden death may take place from heart failure during this stage, or if the patient live longer, death arises from a combination of septicæmia and exhaustion due to pain, loss of sleep and starvation. Later, septic absorption with peritonitis caused by the escape of bacilli through the damaged intestinal wall, or perforation of distension ulcers, may cause death. The latter is rare. A more usual termination is by peritonitis due to the perforation of ulcers in the neighbourhood of the seat of obstruction.

Strangulation by Bands or Internal Hernia.—The most acute cases so closely resemble strangulated hernia that the prognosis in both may be said to be the same without operation.

Intussusception.—In acute cases recovery without surgical intervention is more common than in other varieties of intestinal obstruction.

In children under one year of age the prognosis is worst. They often die in a few hours with all the symptoms of profound shock; in older children and adults death usually occurs in the second and third weeks of the illness. An adult female under my care, with all the symptoms of intussusception, with fæcal vomiting of three days' duration, and a tumour in the position of the ascending colon, suddenly recovered with the disappearance of the tumour, and there is in recorded cases considerable justification for the belief that spontaneous reduction may occur. This, however, must be regarded as an unusual result. The common cause of spontaneous cure is gangrene of the intussuscepted bowel. Gangrene may affect the whole or only part of the invaginated bowel. In a child I saw after it had been ill for a week with symptoms of intussusception, there were several inches of gangrenous intestine with the ileo-cæcal valve at its extremity hanging out of the anus.

The obstruction, as would be expected, is most complete and sudden in acute cases, as in them the vessels of the involved mesentery are at once constricted, and large portions of the gangrenous but intact intestine may be passed per anum. In more chronic cases destruction may be less rapid and more partial. Instead of large areas of gangrene, smaller sloughs of the invaginated intestine may be voided, which may be difficult to recognise as portions of the intestine when passed per anum.

In adults the possibility of polypi as a cause of intussusception has to be remembered, and in elderly persons the still more frequent association with cancer must be borne in mind in making the prognosis.

In the very young, spontaneous elimination by gangrene is almost inevitably fatal.

In youth the prognosis is best; after twenty it becomes steadily worse.

Collected statistics show that out of one hundred cases of intussusception fifteen will recover after spontaneous elimination by gangrene of the intussuscepted bowel. It seems probable, however, that these statistics give too favourable a record for spontaneous cure by gangrene, as recoveries are more likely to be reported in consequence of their interest and rarity than the more usual fatal results.

Spontaneous elimination is usually attended by foul stools containing some blood and masses or shreds of gangrenous intestine, and is followed by diarrhœa. It can only end in cure when the ensheathing and entering layers of gut are firmly glued together by adhesions. The most common cause of a fatal termination is perforation at the neck of the intussusception, and from imperfect union by adhesion of the opposed surfaces. This latter leads to a general peritonitis, or to local peritonitis with abscess, and though the illness in the latter case is usually more prolonged, the ultimate result is the same. Other patients die because the surface at which separation has occurred does not heal, and remaining ulcerated causes diarrhœa, or the ulcer eventually perforates, or causes fatal hæmorrhage from the opening of a mesenteric vessel, which may also occur during the separation of the dead part from the living, or the ulcer produces the different forms of septic disease by absorption of septic products from the raw surface, or as a remote result stricture of the gut may follow the cicatrisation of the ulcer.

Volvulus of the Sigmoid Flexure.—The rapidity with which distension of the bowel occurs and adhesions follow at its neck would make it very unlikely that spontaneous recovery would occur in this condition. Death has occurred a few hours after the onset of symptoms, apparently from shock. Septic absorption from the damaged intestine is the most frequent cause of death, and next to this comes peritonitis. Sudden death from heart failure is even more common in this form of intestinal obstruction than in others, and death from one or other of these causes is almost inevitable during the first week unless the condition can be remedied by operation.

Gall-stones.—About 50 per cent of cases with definite obstruction symptoms due to this cause die during the first ten days. The causes of death are chiefly changes in the intestine above the obstruction leading to septic absorption and peritonitis. Patients who do recover have to pass through a more or less severe illness, followed by evacuation of the gall-stone.

The prognosis of all acute cases of intestinal obstruction taken together is probably a recovery rate of about 15 per cent.

Chronic Intestinal Obstruction

Stricture of the Small Intestine.—In simple stricture the symptoms may be of very long standing—several years, in cases where great attention has been paid to details in matter of diet, and to the regulation of the evacuations. Such a chronic course is only made possible by compensatory hypertrophy of the intestinal walls above the obstruction. In some instances the intestinal walls may be as thick and as strong as those of the stomach. In one of my cases the small bowel reached the size of an ordinary forearm, and served as a receptacle for immense quantities of semi-digested food.

In cases of malignant stricture affecting the small intestine, it is rare for any relief to be possible except through palliative operation.

Spontaneous relief in both simple and malignant cases may occur from ulceration and abscess above the obstruction, if the abscess opens externally and forms a fæcal fistula; it may also, though more rarely, occur from the formation of an anastomosis between the intestine above the stricture and that below it by a fortunate combination of ulceration, adhesion, and perforation. Except for these extraordinary and rare chances the course of intestinal stricture is to a fatal termination. Death is generally brought about by the supervention of an acute attack produced by some object, such as a solid fæcal mass, or a grape-skin or fish-bone, or a cherry-stone, completely obstructing the small lumen of the strictured part of the bowel, or by a kink at the narrow portion. It may also result from malnutrition, caused by interference with the due performance of the digestive and absorptive functions, or it may be brought about by peritonitis, usually due to perforation.

Stricture of the colon always ends in death, but life may be considerably prolonged by a suitable diet and laxative medicines. Many cases have been recorded in which persons with stricture of the colon lived for several weeks without any intestinal evacuation.

The average duration of symptoms is under one year. The average duration of life in cancer of the colon is fifteen months, allowing that palliative operation (colotomy) has been performed for the relief of the obstruction.

The causes of death are the supervention of an acute attack, general peritonitis due to ulceration with perforation above the stricture, or local peritonitis followed by abscess and septicæmia, or gangrene of the stricture from blocking of its vessels of supply by growth in the mesentery. Gangrene causes perforation, and death is brought about by peritonitis.

Spontaneous relief may follow ulceration and the formation of an artificial anus above the strictured part. This may open externally or into the bladder, or into the vagina of the female. In a more rare case upon which I operated for obstruction due to malignant stricture of the sigmoid, gangrene of the entire cæcum and the greater part of the ascending colon from extreme distension was found on opening the abdomen, and the patient was relieved for some weeks by the natural colotomy resulting from the separation of his gangrenous cæcum and ascending colon. Relief may also follow the formation of a natural anastomosis of the intestine above the stricture with that below.

After colotomy in cancer cases death arises most commonly from cancerous involvement of the peritoneum or of the liver; less frequently, from secondary growths in other organs; more rarely it follows sloughing of the diseased part due to blocked mesenteric vessels, followed by localised abscess or general peritonitis.

In chronic intussusception the prognosis is grave. Spontaneous elimination is very rare. After a month of illness, with no sign of spontaneous elimination, death is almost inevitable without surgical operation. The fatal termination generally arises from malnutrition, due to interference with digestion and the absorption of food, or from the supervention of an acute attack, or from perforative peritonitis.

In fæcal accumulation death is very rare, except in lunatics, because the treatment usually adopted proves effective. Death may occur from malnutrition, or from perforation, or from rupture of the distended intestine, the cæcum or the sigmoid flexure being the part to give way.

TREATMENT

The treatment of intestinal obstruction has been made uncertain by difficulties in diagnosis and prognosis, and because both are vague the choice of treatment has been wanting in decision. It must at once be admitted, for every one who has had experience in this disease knows it, that the most unpromising case, in which the symptoms are so marked that a diagnosis of obstruction by a mechanical obstacle is indisputable, may recover spontaneously or under medical treatment.

We also know, however, that treatment by the most approved medical methods, and carried out completely from the onset, results in an appalling mortality; and the same may be said of operations performed as a last resource. Available statistics are useless as an aid in our endeavour to formulate the best methods of treatment. There are no figures to show what is the mortality of operations performed during the first thirty-six hours of the illness, and this should be the extreme time limit if operation is to have the best chance. My opinion, based upon the pathological causes of intestinal obstruction and the general results of abdominal surgery, is that the recovery rate would reach 70 per cent at once if the rule to operate early were universally adopted. In support of this opinion, it may be well to state that of 8 acute cases of intestinal obstruction operated upon during the past year of less than fifty-three hours' duration, in which I have been concerned, 6 have recovered and 2 have died.

Before considering in detail the treatment to be adopted in a case of acute obstruction, I cannot too urgently impress the need for an exhaustive examination, and the formation of the best possible diagnosis, at any rate as to the dangerous character or otherwise of the illness before treatment is commenced.

The most urgent symptom, and the one which always demands the earliest possible relief, is pain. Opium in one or other of its forms is the only drug known which can be relied upon to do this safely, effectually, and speedily. Considering the condition of the stomach and upper intestine, it would appear to be unwise to administer opium by the mouth, as it may be ejected in whole or in part, or, if retained, may fail to become absorbed till, perhaps, the obstruction is relieved, when its effect is not desired, and when indeed a poisonous quantity may find its way into the circulation. The rectum is a better channel for its administration, when it can be given as a suppository or in a small enema; but the best, because the surest and quickest, method is to give a dose of morphia hypodermically. Too large a quantity is to be avoided as hurtful, and one too small, as it will fail to give relief, and may require repetition. A third of a grain in an adult may be generally said to be the proper dose. Repeated small doses of opium are to be condemned in all acute abdominal cases. Administered in frequent small quantities, the drug masks all symptoms and leads to a false sense of security; it paralyses the intestines; and it produces a hyperæsthetic condition of the patient very unfavourable to recovery. A reduction of the accustomed dose leads to a miserable condition of physical and mental collapse, and patients under its influence are predisposed to die from shock after an operation. Though it is true that the rest, intestinal and general, brought about by the use of opium may result in a marvellous improvement in the general condition, I doubt whether the curative influence ascribed to it by generations of authorities has any foundation in fact. If a loop of intestine has been ensnared by a band, it appears to be as reasonable to suppose that, by violent peristaltic efforts, it might again escape as that its release was to be aided by keeping it quietly at rest.

It may have been obvious before the administration of opium that the patient was dangerously and acutely ill, and in all such cases the abdomen should be opened without waiting to see what the result of the opiate may be. Others less certainly ill should be seen at short intervals and closely watched. In ordinary cases a large, hot linseed-meal poultice applied so as to cover the entire abdomen, and left on for an hour, has a soothing effect, and the heat of it diminishes shock, from which the patient is probably suffering. If it be removed at the end of an hour, and, after sponging the skin with hot water and drying it, cotton wool be wound round the body, the weight of the poultice can scarcely be seriously objected to. During the first hours nothing whatever should be swallowed except a few sips of water as hot as can be tolerated, and this should be limited to as small a quantity as the patient's craving for drink will allow. At the end of eight hours, as the effect of the narcotic begins to wear off, if the symptoms return operation should be advised. When the symptoms, then, are not sufficiently definite, and the diagnosis still remains doubtful, my practice is to order calomel in one-grain doses every hour until five grains have been taken, and after the fifth dose to administer a turpentine enema (one ounce of turpentine in a tumblerful of barley-water), followed by one pint of soap and water. If the case is one of obstruction, the symptoms, which are likely to have disappeared soon after the opiate has been given, will probably all be reproduced by this treatment, and the best chance—that given by early operation—is secured. If the case is not one of obstruction this treatment is likely to be the best available, for the cases most closely simulating acute intestinal obstruction are those of so-called colic which occur in young men from eighteen to twenty-five, and are due to chronic constipation and partial retention of fæces.

If the case is not seen until the abdomen has become distended, and the symptoms have all been rendered less aggressive by repeated doses of opium, the question of operation has lost much of its urgency. The condition of the patient may be very serious, but operation may not afford the best chance of recovery. I have seen such seriously ill persons recover under medical measures who, so far as I could judge, would have died had they been subjected to the shock of operation.

The most favourable cases for recovery, without operation, and they only should be left alone, are those in which the obstruction is not absolutely complete.

A little flatus may have been passed, and occasionally peristaltic movements may be perceived in the distended intestines.

The contents of the stomach are expelled with vigour, and vomiting gives relief; quantities of nourishment can be retained and absorbed.

The distension is not so great as to produce difficulty in breathing or marked tension of the abdominal walls, and is not increasing.

The pulse is not growing more rapid or its vigour decreasing.

In the less acute cases, when vomiting has ceased, small quantities of liquid nourishment, half milk and half barley-water, probably being the best, may be tried; and if this is tolerated by the stomach, additions may be carefully and gradually made as the conditions allow.

The three most material palliative aids to recovery are:—Washing out the stomach, large nutrient enemata, and repeated small doses of opium.

Persons differ much in their tolerance of stomach lavage, and it must be allowed that occasionally persistence with it may do more harm than good. The tube used should be a red rubber one, which the patient is assisted to swallow, and attached to it should be a glass T-piece to act on the principle of Sprengel's pump, as neither the efforts of the distended stomach nor the aid of ordinary syphon action may be sufficient to empty the stomach of all its contents. The washing should be done with water at a temperature of 102° F., and continued until the wash water returns clear. If this operation is to be of service the patient must feel relieved by it, and will consider the gain greater than the discomfort to which he has been subjected, and this should be the test as to its usefulness and as to the desirability of repeating the proceeding. If the use of the tube is found to be too disagreeable, the best substitute is to allow the patient an occasional hot drink (a pint of very weak hot tea without sugar or milk is nearly always to his taste). This is soon ejected, bringing with it a quantity of the foul stomach contents. Very soon after the stomach is emptied it commences to refill from the discharge of the distended intestine into it, and the washing requires to be repeated every four hours.

Inability to bear the tube should be regarded as indicating the advisability of early operation.

No food should be allowed to enter the stomach so long as sickness is a prominent symptom. Food and fluids introduced into the stomach only add to the sufferings of the patient, and often diminish what small chance of recovery is left to him. To relieve the intense thirst sips of hot water may be frequently given, and the mouth may be sluiced round with cold water as often as desired by the patient. Ice gives only temporary relief, and does permanent harm. It makes the mouth sore and the tongue dry; it fills the stomach with cold water, and increases flatulent distension.

All nutriment should be administered by the colon. The best method of doing this is to give slowly through a rectal tube and raised funnel one pint of a mixture of fatty beef-tea and milk in equal parts, with one teaspoonful of salt and half an ounce of whisky at a temperature of 100° F. every two or three hours. The first two or three pints are frequently retained when given in this way, whereas the intermittent pumping action of the ordinary enema-syringe commonly used for this purpose stimulates the bowel to expel the injected fluid. When the enemata begin to come away, it is, of course, a favourable omen if some flatus is passed at the same time. Diminution of the abdominal distension, as shown by careful measurement round the umbilicus, is also a favourable sign. If the enemata are retained for twenty-four hours it is necessary to administer a quart of salt water to clear out the offensive débris, as in all cases where food is given by the bowel. From the use of nutrient suppositories little good need be expected, and if the enemata cannot be retained their expulsion is a strong indication for early operation. In connection with enemata, it may be mentioned that their use is strongly recommended by nearly all authorities in the treatment of intussusception. It is agreed that they have no value in the most acute cases or in cases involving the small intestine. It is also agreed that there is a certain amount of danger in their use. Before giving the enema, if this is decided upon, an anæsthetic must be administered to complete relaxation. Hot salt water, a drachm to a pint, is the least irritating injection, and as much of it as can be retained should be introduced. The enema funnel should not be raised more than three feet in the case of children, who may be held up by the feet during the administration. It is usually impossible to say when this operation is completed, even when a tumour can be distinctly felt and disappears, that reduction has been accomplished, and valuable time may be lost in waiting to see if the symptoms return. It is usually safe to prophesy that they will do so, and, except in the more chronic cases, in which a short period may be allowed to be wasted with comparative impunity, operation ought to be done as early as diagnosis permits.

Repeated small doses of morphia in the favourable cases previously mentioned are often of great service—10 ♏ of liq. morphia hydrochlor. in one drachm of peppermint water, to be given by the mouth, when the patient is restless or in pain, secures bodily and mental rest, and has a stimulating effect on both mind and body.

If nothing short of narcotism brings relief, operation is urgently indicated.

In addition to the use of stomach lavage, nutrient and stimulating enemata, and morphia, which are the most important means, there are smaller details requiring attention. A hot, moist poultice, large enough to cover the whole abdomen, kept on for an hour and repeated at intervals of eight hours, and replaced by warm cotton wool, is useful. A suitable bed and a good nurse are great comforts, and are important aids to recovery.

Operation in Acute Intestinal Obstruction.—In cases of acute intestinal obstruction the sooner operation can be performed the better the prognosis. It is, indeed, now amongst surgeons an established rule that there should be no unnecessary delay in opening the abdomen for any cause producing sudden alarming symptoms, and the sooner this rule is applied to cases of intestinal obstruction the better. That the dangers of delay far outweigh the dangers of operation, serious though these may be, is the most important point to bear in mind in connection with the treatment of intestinal obstruction. During the first forty-eight hours it is usually possible to perform an operation which allows the cause of the obstruction to be dealt with, and which offers good prospects of complete success.

Preparation of the Patient for Operation.—As soon as excruciating pain has been relieved by morphia, and the symptoms of collapse have subsided, the skin of the patient is prepared as for an abdominal section. It is of the greatest importance to have a warm room for the operation, and by hot bottles and warm clothing to prevent chilling of the patient on the table. The arms and legs should be swathed and bandaged. Children especially require exceptional trouble to be taken in this respect.

Any special instruments, such as intestinal clamps, Murphy's buttons, Paul's tube, should be ready for use, and everything should be prepared before an anæsthetic is administered.

Chloroform in the hands of a skilled administrator is the best anæsthetic for general use. Before its administration the patient's stomach should be washed out, to avoid the risk of regurgitating septic fluid being drawn into the lungs.

Abdominal palpation should be repeated under the anæsthetic, as a tumour which has previously escaped observation may now be discovered.

The incision is commenced one inch above the umbilicus, which is included in an ellipse, and prolonged to two inches below it. The umbilicus is then excised. Excision of the umbilicus removes that portion of the abdominal wall which is most difficult to disinfect. It allows of safe extension of the wound upwards and downwards, avoids the chance of damage to the bladder, and renders access to the abdominal cavity through the umbilical ring easier and safer than any other situation. The incision is then continued through the linea alba upwards and downwards from the umbilical ring, and the peritoneum is drawn forward between catch forceps away from the underlying viscera, and is opened. *The first object of the operation*—to confirm the diagnosis—will now be realised, for from the earliest stage the intestine above the obstruction is observed to be distended and increased in vascularity, and inflammatory peritoneal exudates are absent. The intestines are carefully kept inside of the abdominal cavity by a warm flat sponge or sterile swab, and a careful search is made in the neighbourhood of the wound, which should be opened by retractors. If nothing be seen there, two fingers should be gently passed in for exploratory purposes, and the most vascular and distended coils of intestine, which, it will be remembered, are above the obstruction, and which may be recognised as belonging to either the large or the small intestine, are gently pushed aside. *The second object of the operation* has now been attained, and the obstruction localised in the small or the large intestine. Supposing the small intestine to be affected, and a pale and contracted loop, which points certainly to bowel below the obstruction, be found, it should be drawn forward and followed inch by inch until it guides to the seat of obstruction or to its termination in the ileum. In the latter case a journey will have to be made in the opposite direction, and this leads to the obstruction. Each portion of the bowel is carefully returned to the abdomen or protected by hot towels as it is passed through the fingers. The third point to be attended to in the operation, of finding the obstruction with as little disturbance or handling of the distended and friable intestine as possible, has now been achieved.

If the obstruction is not readily discovered in this way the abdominal incision should be prolonged to just above the pubis, taking care to see and to avoid the bladder, and the exposed intestines are to be covered with aseptic towels wrung out of hot normal saline solution. Any uncovered portion of intestine should be kept hot and moist by an assistant steadily dripping hot saline solution (℥j. to 1 pint) over it and the surrounding coils and protecting towels from a jug during the whole time of exposure. Commencing on the right side, the condition of the cæcum, dilated if the obstruction is below it, will tell whether the obstruction be in the large or in the small intestine. If it is in the small intestine the contracted end of the ileum entering the cæcum may be followed up to its junction with the distended bowel above, and so discover the site of obstruction.

If the obstruction be localised in the large intestine, recognised by its longitudinal bands, the sigmoid flexure is first drawn up for examination, and if this be vascular and distended the obstruction will be found towards its lower end. If not, the transverse colon should be brought forward by drawing on the omentum, on the under surface of which this portion of bowel will be found; finally, the ascending colon and cæcum are examined.

As a last resource, by an extension of the abdominal incision to the ensiform cartilage and evisceration, any obstruction can certainly be found.

Evisceration adds greatly to the gravity of the operation, and should only be undertaken in vigorous subjects not reduced by long illness. It is to be preferred to rough handling of the distended and friable intestine, but, as a rule, the best proceeding at this stage is to draw out and to open a coil of tense intestine, for in any case the distended intestine must not be replaced in the abdomen. The most tense intestinal coil visible should be drawn forward well out of the abdomen, the opening into which should be isolated with aseptic gauze packs. When the extruded coil is thoroughly shut off from contact with the wound, it is opened by a longitudinal incision about half an inch long through the wall opposite to its mesentery, and the intestine is allowed to empty itself. If it does so without help, pumping out its contents vigorously, and the general condition of the patient is fair, after temporarily closing the intestinal opening with clamp forceps, a further search for the cause of the obstruction should be made, and this is rendered more easy by the diminished abdominal tension. If the obstruction is now discovered, and can be relieved, after careful suture and cleansing of the intestinal wound, the abdomen may be entirely closed. If, however, the intestine, though obviously distended and tense, refuses to empty itself without aid, and if, after a pinch between the finger and thumb, no peristaltic movement is seen in it, enterostomy offers the best chance of recovery, for if the paretic intestine is subjected to further manipulation neither it nor the patient is likely to recover.

If the obstruction is found to be an intussusception, and if it is acute, and the intussusception is swollen and firmly strangulated, and there are no adhesions, the surgeon's first duty is to lessen the œdematous swelling of the intussusceptum which is the chief barrier to reduction. The tumour is to be drawn forwards, wrapped in a large, warm, flat aseptic sponge, and gently but firmly squeezed by gripping it in the hand. The pressure must be continued till the swelling has subsided, then an attempt should be made to reduce the intussusception. This is done best by a combination of kneading from below and gentle traction from above, most reliance being placed on pushing from below, as it is easy to tear the intestine by drawing on it too freely. Adhesions may prevent reduction. These can be separated by a blunt director introduced between the ensheathing layer and the intussuscepted portion of bowel. If it is not possible to effect reduction, and the intussusception is of limited size, the best course is to excise the whole of the involved intestine. The abdominal cavity all round the intussusception is packed off with aseptic gauze, and the portion of intestine to be excised is completely isolated by this means. Two pairs of clamp forceps are fixed above the involved portion of intestine and two below. The intestine is divided between the two clamps above and below, and the intussusception is isolated. Then by gradually snipping through the mesentery with scissors, each vessel being secured before division if large, and caught as divided if small, the mass is separated. After ligature of the clamped mesenteric vessels and the arrest of all bleeding, the treatment of the divided intestine has to be decided upon. This depends upon the condition of the patient. If it is good, the continuity of the intestinal canal must be restored by an end-to-end or lateral anastomosis, of which I prefer the latter. If the condition of the patient does not allow of this, the involved portion of intestine must be drawn out and packed round with gauze in such a manner as to thoroughly isolate it and block the abdominal wound; then the bowel is to be opened above the intussusception and slit up through the length of it to relieve the strangulation and permit of fæces passing through the artificial anus. This operation, if the patient recovers, requires a second one at a later date to restore the continuity of the canal, and this is a disadvantage, but the present safety of the patient must be the first consideration.

Volvulus.—After the abdomen is opened, the marked distension and vascular disturbance of the sigmoid flexure, easily recognised by its longitudinal bands and appendices epiploicæ, which become apparent, makes the diagnosis clear. An attempt should be made to guide a tube introduced through the anus into the distended bowel for the purpose of emptying it. If this cannot be done the patient should be turned over on to his side, and the wound, being protected by gauze, the presenting loop of sigmoid should be incised longitudinally and drawn forward by a pair of hæmostatic forceps fixed on either side of the incision. As the contents escape, the bowel is to be further drawn out from the wound by the forceps. When empty of its contents the bowel is washed, wiped dry, and the opening in it is closed with clamp forceps, the whole being covered with iodoform gauze. An examination of the empty sigmoid loop is then undertaken and the twist undone. The hole, if it has been necessary to make one in the bowel, is next closed with a continuous suture of catgut through all its coats, with the object of making it watertight and arresting all bleeding. This first row is covered in by a second row of fine silk Lembert sutures, and the bowel is cleansed and returned. Before closing the abdomen the centre of the loop should be sutured to the parietal peritoneum to prevent a relapse.

Bands.—As these mostly contain a blood-vessel, and are sometimes a hollow prolongation of Meckel's diverticulum, it is a wise precaution before dividing them to apply two clamps and cut between, subsequently ligaturing each end. The portion of bowel nipped by the band should be examined before letting it drop back into the abdomen. I lost a patient on one occasion by perforation of the intestine at the ulcerated site of a narrow band on the tenth day after operation. If the ring of the intestine does not recover its normal appearance after the application of a hot sponge, it should be doubled in by drawing the intestine above and below over it, fixation being secured by a few fine silk Lembert sutures. If then it should slough or ulcerate, it will do so safely into the lumen of the gut.

Internal Herniæ.—If a loop of the bowel ensnared cannot be withdrawn by the opening through which it has passed, without enlargement, the frequency with which a blood-vessel surrounds this opening has to be borne in mind. If possible, the safest method is to enlarge the aperture by stretching with sinus forceps passed through it guided by a director. The same precaution as to examining the nipped intestine is necessary in this case as in that of a band.

Cases of hernia into the duodeno-jejunal fossa have been recorded in which no portion of the small intestine was visible on opening the abdomen, the whole of the jejunum and ileum having passed into a pouch, of which this was the neck. A knowledge of this fact will make a recognition of the condition to be dealt with and its treatment easier.

Gall-stones.—A gall-stone large enough to cause obstruction will probably be readily recognised by intra-abdominal palpation. The portion of intestine containing it should be drawn forward and examined. If the stone is lodged close to the ileo-cæcal valve, a gentle attempt should be made to push it on into the colon, where it may be left to pass on by natural efforts. If this cannot be done, or if the stone is lodged in the jejunum, or higher in the ileum, it should be squeezed upwards for some inches into an unirritated piece of intestine; and after this has been drawn out of the abdomen, the intestinal coats over the stone should be incised longitudinally at the convexity of the intestine, emptied of the stone and other contents, and sutured in the manner previously described, and returned with the precautions for avoiding sepsis already mentioned.

Stricture of the Small Intestine.—If the stricture is simple and annular it is readily and safely dealt with by enteroplasty. In this operation the intestine is opened three-quarters of an inch above the stricture on its convex side, and a guide is passed on through the stricture, which is divided in the same line by an incision extending three-quarters of an inch below the stricture. The longitudinal wound is cleansed and sutured transversely with an internal row of catgut sutures passing through all the coats, and an external row of silk Lembert sutures burying it. The same treatment is also occasionally the best palliative for annular malignant stricture of the small intestine which is not removable as such strictures usually are, but as a rule in these cases an anastomosis above and below is the most satisfactory method of treatment. More complicated strictures, as for example those associated with active tuberculous disease, are best treated by excision of the diseased and strictured portion of the intestine, or by making an anastomosis above and below the stricture. If the condition of the patient does not allow of this, enterostomy above the stricture must be performed as a palliative measure.

Stricture of the Large Intestine.—If the patient is vigorous enough to allow of exploration, the operation should be conducted with two objects in view—the remote one of a radical cure, and the immediate one of giving relief. This exploration, which should be accomplished in a few minutes, should demonstrate for future guidance the exact situation of the disease; its character, simple or malignant; if malignant, whether fixed or movable; and the presence or otherwise of enlarged glands or secondary deposits in the liver and elsewhere.

It is seldom wise to attempt a radical cure and relief of obstruction in the colon at the same time, for wounds in the colon do not unite so readily as those in the small intestine, and are, in addition, exposed to traumatism by the passage of solid fæces. Therefore colotomy should be first done as immediately above the obstruction as seems advisable, and the radical operation left for a later date.

The complete operations we have described as applied to obstruction affecting the small intestine are only possible with a fair chance of success when diagnosis has been made sufficiently early, and surgical treatment has been carried out at once. Patients with acute intestinal obstruction are even worse than they look, and seldom get well after a complete or prolonged operation, however satisfactory, from the operative surgical point of view, if the small intestines are considerably distended. Such an operation, even though it empties the distended intestine, completes the intestinal paresis already commenced, and the patient gets relief after operation only for a few hours.

The operation of choice for such patients is enterostomy. It has been my misfortune to perform this operation, and to find on subsequent post-mortem examination an intestinal opening 12 inches below an impacted gall-stone lying immediately under the anterior abdominal wall; to discover after an immediately successful enterostomy that death on the ninth day was due to gangrene of a loop of bowel ensnared by a string-like adhesion easily accessible, and to see curdled milk oozing through the fistulous opening soon after it had been swallowed, showing that the incision had been made so high in the jejunum that death from starvation must shortly be expected. Against these disappointing results, which a more thorough surgical procedure could have averted only by the certain sacrifice of the patient's life, must be placed the cases in which enterostomy has afforded another chance of life, which the more complete operation did not. A second operation is generally required after enterostomy, but it is not a unique experience to find the symptoms permanently relieved, and to see the patient completely cured, with spontaneous healing of the fæcal fistula.

Enterostomy may generally be regarded as a protest against delay, but until early operation becomes more frequent it holds a useful place in the treatment of intestinal obstruction.

The operation is best done by an oblique incision from above downwards in the direction of the fibres of the external oblique muscle about three inches long, commencing in the right side at the centre of the back of the ileo-costal space and extending as far forward as needful. The cæcum, if distended, appears in the wound, is drawn outwards and opened towards the posterior extremity of the wound, after this has been packed thoroughly with protective gauze. A Paul's tube is then tied into the bowel, the ligature and the junction between bowel and tube are wrapped in iodoform gauze, and the parietes are sutured with silkworm gut above and below, the two sutures nearest the intestinal opening coming through the outer coats of and fixing the gut.

If the cæcum is not distended the nearest coil of tense small intestine is drawn out and pulled towards the back, opened, and similarly treated.

The advantages of this incision over one in the middle line are that it exposes a favourable site for pathological conditions, the neighbourhood of the ileo-cæcal valve; that if the large intestine be at fault, typhlotomy can readily be done; that there is less difficulty in retaining the intestines in the abdomen with this than with a median incision; that if the small intestine has to be opened, it is here that the ileum, not the jejunum, is likely to be found; and that by keeping the intestinal opening posterior there is less risk of the entrance into the abdomen of its septic contents. This operation can be done with local anæsthesia alone, but it seems better to combine the use of a local with a general anæsthetic. If the skin be frozen with chloride of ethyl, a skilled chloroformist with very few drops of chloroform can dull consciousness enough to render the patient comfortable and incapable of feeling pain or being fearful.

If the fæcal fistula left by this operation shows no tendency to close spontaneously, the abdomen should be opened in the middle line during the third week or thereabouts for the purpose of treating the cause of the intestinal obstruction, and at the same time or later, as seems best, the enterostomy opening can be closed by a plastic operation.

After-treatment of Operation Cases.—The first indications in the after-treatment are to relieve shock and procure rest. To treat shock the patient should be heated in bed by warm bottles and blankets all round, and an enema of two ozs. of beef tea, two ozs. of milk, and half oz. of whisky should be administered. His face and neck and hands should be rubbed with a warm towel and one-twentieth of a grain of strychnine given hypodermically. If recovery does not soon manifest itself, one pint of normal sterile saline should be slowly injected under the pectoral muscle on each side, with strict precautions against septic contamination. For the purpose of procuring rest a single dose of morphia, one-third of a grain hypodermically, is ordered as soon as the patient begins to complain of pain or to get restless. In twelve hours five administrations of calomel in one-grain hourly doses should be commenced unless flatus has been freely passed, when nothing should be given. After five doses of calomel, if flatus still refuses to come away, the insertion of a rectal tube may be tried. If this fail, a turpentine and soap-and-water enema will assist it, and this may be repeated every four hours. Opium should not be given after the first dose unless the patient is obviously dying.

In the matter of feeding, my rule is not to allow more than the smallest quantity of hot fluid, usually a teaspoonful of hot water with a few drops of brandy in it, if required, until flatus has been passed, then to administer small and gradually increasing quantities of milk and barley water. As soon as free evacuation of the bowels has occurred more solid food may be partaken of.

Intubation.—The operation of intubation or tubage of the larynx consists in the introduction through the mouth of a metal tube provided with a collar which, when the tube is in position, lies upon the vocal cords. The tubes are of different sizes, and are graduated according to the age of the patient. The introduction is effected by means of an *introducer*, an instrument consisting of a long curved stem and handle, and provided at the end with a screw or catch to which is fixed the jointed metal guide on which each tube fits loosely. A gag having been inserted in the left side of the mouth, the patient may be held in a sitting posture on a nurse's knee, or may be placed lying on the back with the limbs firmly controlled. The operator introduces the left forefinger, and passes it behind the epiglottis. As soon as he feels the opening of the glottis he introduces the tube alongside his finger, and lifting the handle of the introducer, he brings the point of the tube under the tip of the finger and into the opening of the glottis. Then by means of a mechanism which differs in the different instruments he pushes the tube off the guide and leaves it lying in position in the larynx.

As the operation is one of considerable difficulty, and there is always a chance of the tube slipping into the œsophagus, it is absolutely necessary that a loop of thread be passed through a hole in the collar of the tube, so that if the tube is not in position, it can be at once withdrawn. It is usual after the operation is completed to remove this string by pulling on one end, but some prefer to leave it, and fix it by sticking plaster to the outside of the child's cheek. The advantage of this method is that should the tube be suddenly blocked by a piece of membrane, a nurse can pull it out at once, instead of having to wait till the surgeon uses the extractor provided with every set of instruments. The patient readily accustoms himself, moreover, to the presence of the string in the mouth. It may be added, that if the string is to be left in permanently, wire banjo string is most suitable as not being so easily bitten through.

If the obstruction for which the operation has been performed is at, or in the immediate neighbourhood of the glottis, the introduction of the tube relieves the symptoms at once. There is usually a certain difficulty in swallowing at first, but in the hands of careful nurses, if the patient is fed sufficiently slowly, it is easy to supply an adequate amount of nourishment. Soft solids, jelly, baked custard pudding, and so on, are taken better than fluids. Nasal feeding, if necessary, can be resorted to.

If there are no marked signs of irritation and no aggravation of the temperature, the tube may be left in from two to three days, when it should be taken out and cleaned. Quite frequently it is unnecessary to replace it. In any case it should not be reintroduced unless the state of the patient absolutely requires it.

Advantages.—The advantages of this operation are that it may often prevent the necessity of a cutting operation, so dreaded by parents, and that it, in the case of diphtheria, gives time for antitoxin to stop the spread of the disease without exposing the patient to a tracheotomy. The *disadvantages* are unfortunately more numerous. In the first place, it cannot be said to give rest to the affected part. Secondly, the tube may push down membrane before it, and so inoculate the larynx lower down. Thirdly, if the membrane is loose, it may be even rolled up in a ball by the introduction of the tube, and absolutely plug the larynx below the point of the instrument. And although these objections which are largely theoretical may be discounted, it is impossible to dispute that the operation is totally unsuited for general practice. In the first place; constant practice is required if the operator is to give his patient every chance. Secondly, a nurse cannot replace a coughed up tube as she can that of a tracheotomy, and as this accident frequently happens, unless a doctor can be always on the spot the patient's life is not safe.

In infectious hospitals, however, where a large amount of diphtheritic croup is treated, and where there are resident medical men, the introduction of antitoxin has given a new lease of life to intubation as an operation. It is always worth trying in the first place, and if it does not relieve the patient, tracheotomy should be done at once without waiting too long. It is quite unusual nowadays to see membrane spread after antitoxin has been given in adequate doses, and therefore the objections regarding the inoculation of healthy parts of the larynx no longer hold. There is no reason why an operation which is so popular in America and France should not give good results in hospital practice in this country.[1]

Intussusception. *See* INTESTINE.

Inunction.—Inunction consists in the application of a medicinal substance to the skin in such a manner as will lead to its absorption. Various diseases, both of a local and general nature, may be treated by this method. Of these, perhaps the best illustrations may be found in the use of mercury for syphilis, cod-liver oil or other fatty substance, in cases of markedly impaired nutrition, and olive or other oil in certain cases of chronic rheumatism. The beneficial effects accruing from such use of oils are not sufficiently appreciated. In the cases that are materially improved by this treatment the benefit is probably explained by the influence of the oil on the local, and it may be general derangement of fat metabolism in the body. No matter what the nature of the case may be there are certain elementary points to be attended to. These can be stated shortly:—(i.) A preliminary stimulation and softening of the skin by heat in the form of hot water immersion is of great service in furthering the efficient absorption of the drug.

(ii.) The oily fluid or the ointment should be thoroughly rubbed into the selected part at bed-time, the friction being mainly from below upwards, and continued for ten to twenty minutes.

(iii.) Thereafter the limb or other part of the body should be covered with a layer of flannel more or less impregnated with the substance, and left in contact over night. The parts are thoroughly washed in the morning, and the treatment continued at the discretion of the physician.

Lately additional interest has been given to this subject by the strong expression of opinion by various writers to the effect, that, so far as mercury is concerned, the system becomes impregnated with it, not by a process of cutaneous absorption, as has long been held, but by a process of volatilisation. Recent clinical experience, it may be added, lends a good deal in support of this belief. The methods recently introduced consist in applying to the surface of the chest, as a kind of under vest, a specially prepared cloth containing a mercurial preparation in its interior. The advantages of this manner of administration from a point of view of cleanliness are apparent, and if a more extended experience confirms its efficiency, the older system of inunction will probably give place entirely to it.

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[1] The usual instruments used are those of O'Dwyer. They have been modified by Colin, whose tubes are shorter and whose introducer is more easily kept clean.

Invalid Feeding.

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See also DIET, and FOOD (PUBLIC HEALTH, ASPECTS OF).

INTRODUCTORY.—The nutritive constituents of food, their relative values, and the factors which modify individual requirements in health, have already been considered in the article on "Diet" (vol. ii.) In disease, as in health, we must have regard to the functions of food, as *building material*, *fuel*, and *suppliers of energy;* and we have here also to consider the modifying influence of age and sex, height and build, and the amount of work or rest to be indulged in, and any personal peculiarities in the requirements of each individual case. But, above all, we must remember that if the palate, or the sense of sight or smell, be offended, no matter what the nutritive value of a food may be, the food is valueless; and as the most frequent cause of offence lies in a want of knowledge of certain practical details in the manner and preparation of foods, the scope of this article will be mainly limited to noting these points. Every practitioner should possess some knowledge of cookery. It is idle for the practitioner to recommend certain food-stuffs without taking all necessary precautions to ensure that the manner of their preparation is suited to the palate as well as to the physiological capacity of the patient. Yet such recommendations are by no means rarely made. While in many houses where there is a thoroughly competent housewife or a trained nurse, any directions regarding the manner of preparation of egg flip, sweetbread, or the cooking of an oyster, may be superfluous, other cases are only too numerous where, if the practitioner has not the practical knowledge to enable him to give his instructions in detail, the patient suffers in consequence. To illustrate: The following instructions for an egg flip are frequently given:—"Beat up an egg; add a little milk, a little sugar, and a stated quantity of brandy." What is the result? For lack of the requisite straining of the mixture the second or third mouthful taken by the patient may include the streaky, tenacious portion known as the trend, which is to many people so nauseating that no more egg flip will be taken at that time or for many a day thereafter. And in the same way with many other dishes.

GENERAL PREPARATION OF FOOD.—Cooking increases the digestibility of food-stuffs by exercising a mechanical, chemical, and antiparasitic effect on the food, and it is of great importance in ordering a diet for an invalid that the physician should understand at least the main principles by which the food is prepared.

The mechanical action is a twofold one: the prolonged heat acts by softening hard tissues and by preparing tough tissues for mastication. Also, as the functions of digestion and assimilation are best carried on at a blood heat, one object of cooking is to raise the food to a suitable temperature, and thus spare the bodily energies as much as possible.

The chemical changes taking place act on the meat substances by transforming the connective tissues into gelatine, the proteids of the meat at the same time developing odorous substances which impart flavour and stimulate the appetite. The starch granules are broken up and prepared for the action of the saliva, and the cooking converts some of the starch into dextrine and even into maltose. These chemical changes can be further carried on by the addition of some of the pancreatic ferments, which transform the albuminous substances into peptones, and also act on the starches, and carry on the further transformation of the starches into soluble sugars. These more complicated changes are further considered under the Predigested and Prepared Food section, see p. 467.

Not the least important action of cooking is the destruction of the parasites and pathogenic bacteria of disease that may occur in the food.

The effect of different degrees of heat on albumin is the keynote to all the various culinary methods, which are variations of one another depending on whether the meat juices are to be retained or extracted.

(*a*) When the meat juices are to be *retained* there is, first, a preliminary case hardening of the albumin by intense heat; after which, cooking proceeds at a lower temperature, rising in the interior of the meat just to the coagulating point of albumin. This object may be carried out by any of the following methods:—

(i.) Roasting, Grilling, Baking.—The heat being radiant heat and hot gases.

(ii.) Steaming and Boiling.—In this the heat is hot water.

(iii.) Frying, Wet or Dry.—This process is carried on by hot oil or hot oil and conduction.

By following these methods we retain all the nutritive, sapid qualities within the portion, leaving none of them, or very little, in the medium used.

(*b*) If the juices are to be *extracted* from the meat the result is a stew or a soup.

Stewing first extracts the juice and then cooks the meat in it, and is thus conducted slowly and over a long time.

Soup-making aims at extracting as much as possible, and therefore begins in the cold, never being above about 160° F.

These various processes will now be referred to in some detail.

A. *When the Meat Juices are Retained*

Roasting.—In this method of cookery the meat is cooked by the radiant heat of a fire from a large glowing surface free from smoke. The meat is exposed to this heat, within a few inches, for 5 to 10 minutes, and is basted with melted dripping, the surface albumin being coagulated to the thickness of a sixpence. After this has taken place the meat is removed ½ to 1 ft. farther from the fire, and the cooking process is continued slowly at a greatly reduced temperature, the meat being slowly cooked in its own juices.

If the heat is too great the hardened case is apt to crack, allowing the juices to escape. To prevent this the meat is basted with hot dripping, which prevents charring and cracking of the surface. If properly done this fat does not soak into the meat. Although the surface heat is very great, the heat in the interior of the joint should not be much above the coagulating point of albumin, and any rise above this renders the meat less digestible, the albuminous matters becoming hard and horny.

The inside parts of a *properly* toasted joint are the best for a weak digestion.

This process requires a quarter of an hour per pound for beef and mutton, but longer for veal and poultry. The meat loses weight from expulsion of water.

Baking.—Joints of meat can be well cooked in the oven of an ordinary kitchen range or in a gas cookery, and the results are very similar to roasting. If the heat of the oven is not properly regulated the results are different, the meat being richer and more indigestible, and it has a different flavour. This is owing to the process being conducted at too high a temperature after the preliminary case hardening.

Broiling, Grilling, or Brandering.—This is a similar process to roasting, and is admirably adapted for quickly cooking a chop, steak, and kidney, fish, fowl, or mushroom, etc.

A clear and smokeless fire is required, and is carried on by holding the article to be cooked over the fire.

The object is the same—the formation of a surface skin of coagulated albumin, and the retention within this of all the juices of the meat. To avoid breaking this coagulated layer steak tongs should be used instead of forks for turning the meat.

In this form of cooking the process is so rapidly carried on that there is not much time allowed for softening the food, so that, unless the meat is very tender, this process is not to be recommended.

Boiling and steaming as described here consists in cooking meat, fish, or vegetables either with hot water or steam, with a view to retain in the meat all the flavours, and is the opposite of soup-making, which aims at withdrawing the meat juices. Here, again, the first step is to coagulate a surface layer of albumin by plunging the joint into a large pot of boiling water, to which has been added a handful of salt. This is done on account of the boiling-point of salt water being higher than that of fresh water. The formation of this hardened case takes about seven minutes; the pot should then be withdrawn and kept at the side of the fire, not even simmering. This method is not only much more economical, but the meat is very much more tender if the cooking is slowly conducted over a period of five or six hours. The best method of slow cooking is by steaming the article of food in its own juice; this can be done most satisfactorily in a Warren cooking pot. This is a pot consisting of three compartments connected by a steam chimney. Water is put into the lowest pot, in the second the meat and its flavourings, and in the top the vegetables. The food is thus cooked by steam; all the juices of the meat are retained; and the meat will be found to be much more succulent, tender, and digestible than if simply boiled in water. If the boiling is carried to excess the meat is "boiled to rags," but this only means that all the connective tissue has become gelatinised, and the muscle fibre remains as tough as leather, and may often be recognised unchanged in the fæces.

The boiling of vegetables aims at softening the cellulose and rendering it tender and digestible; it also opens up the starch granules, so that the saliva can get at them, and converts some of the starch into dextrine. Vegetable proteids, when in the form of legumin, are not coagulable by heat, so overcooking is not so injurious, but merely entails a waste of fuel.

Green vegetables and rice are eaten on account of the salts they contain. Therefore, unless they are steamed, or the water in which they are boiled is kept as stock for soup, the best part of their substance is thrown away. Steaming has the further advantage of preserving the shape of the vegetable or fish, and of therefore presenting it in a more appetising form.

Frying is boiling in oil, but as practised in this country is a dirty and wasteful method, being usually a combination of broiling and scorching. There are two forms. In *dry frying*, which is our national form of frying—a shallow frying-pan is used, and the food is either cooked in its own fat or with the aid of sufficient fat to prevent burning. It is only suitable for fatty food like herrings and sausages, but the products of this form of frying are often greasy, and for many persons exceedingly indigestible.

Wet Frying or sautéing is properly boiling in oil. For this there is required a deep pan of clarified dripping or of olive oil. If the process is properly carried out the food is deliciously cooked and absolutely free from grease. The temperature of the fat is important; when fat is placed on the fire and reaches 212° F. (the boiling-point of water), it bubbles and makes a hissing sound due to a small portion of the water in the fat becoming steam and being got rid off. Fat, however, does not boil until it has reached a much higher temperature. At a temperature of 340° F. a slight bluish vapour is given off, and this is the time when if a piece of bread be dropped in, it becomes brown in a second or two, and on being taken out of the fat, almost instantly loses all its greasy appearance. If the fat gets hotter and begins to boil, a state of affairs to be avoided, it begins to smoke and decompose.

The principle on which the success of frying depends is that at the moment of contact with the almost boiling fat a thin film is formed over every part of the surface of the fish or other object to be fried, and all the juices and flavours are retained. The food is kept in until it has a golden brown colour, when it is removed from the oil bath. It may look greasy for a moment, but this drains off very quickly if put on a piece of paper near the fire, and it is absolutely free from grease when it appears at table. If the object to be fried is coated with egg and bread crumbs the crumbs should be *very* fine, and firmly pressed down to prevent the grease adhering to the surface and by being absorbed by it.

The best substance to use is second quality of olive oil, or roast beef dripping; clarified lard should never be used, as the other media for frying are as easily obtained and are much better.

B. *When the Juices are Extracted*

Stewing and braising are processes by which meat and poultry can be prepared and served in the most tender condition possible.

Stewing aims at making an extract of the nutritive juices of the food which are then used for cooking. A low steady heat is wanted, and since nothing is lost in this process it is one of the most economical ways of preparing food. The meat is chopped into convenient sized portions, seasoned (if there are bones they also are broken up and put into the pot), the whole is covered with cold water, and a tight-fitting lid put on to the pot, which is placed near the fire, but never allowed to boil. The old saying, "A stew boiled is a stew spoiled," is a true one. If vegetables are to be added they are best put into the pot after the meat is half done, The flavours of the vegetables are lost with too continuous cooking. The more slowly the stew is cooked the better it is done. Tough meats are rendered more digestible by the addition of a little vinegar to loosen their fibres and to convert them into acid albumoses.

Braising is slow cooking in a closed vessel, the meat being just covered with an extract of animal and vegetable juices instead of water, and so the flavour of the meat is improved. This method is best adapted for foods somewhat insipid in themselves like veal and poultry. At the end of the process the meat should be browned, and this is carried out by having a concave lid in which hot cinders can be placed, or it may be done by radiation before the fire.

Soup-making.—This, like stewing, proceeds upon the principle of extracting as much as possible from the materials, the extraction being effected by the use of a considerable quantity of water at a very moderate heat extending over a long period.

The simplest form of soup-making is that of making beef tea, page 475.

The meat is cut up into several pieces to expose as large a surface to the water as possible; the solvent power of the water is increased by the addition of a little vinegar, and the temperature must never rise over 160° F.

As vegetables require a very much greater heat than this to soften them, they should either be first boiled, and then the meat should be added; or the steamed vegetables should be added when the soup is almost ready. Flavouring herbs should be put in at the last moment. In all soups made from meat great care should be exercised in the removal of fat. This is best done by the soup being made the day before it is required, and poured into a basin and carefully skimmed when cold.

VARIETIES OF SOUP.—*Stock.*—This is the simplest form of soup, and is the product of the mysterious stock-pot. This vessel should receive all scraps of meat and vegetables that are over from the preparation of other dishes. It stands constantly at the side of the stove, gradually concentrating its contents, and it requires to be brought to the boil every day for five minutes to prevent fermentation. It should also be cleaned out every third or fourth day in case of the stock going sour. This stock can be used for many soups, for the basis of gravy, and, instead of water, in stewing and boiling meat.

The next variety of soup is that which is made on the lines of a good Scotch broth of meat and vegetables either boiled in weak stock or with water; this is known in France as pot-au-feu, and here the boiled meat is intended to be eaten either with or immediately after the soup.

A stronger extract than pot-au-feu is grand bouillon, in which to the meat and vegetables are added bones and connective tissue for the sake of the gelatine. This, when cold and strained, forms a slightly firm jelly, which can be cleared and flavoured in many ways.

A still richer form of soup is one which to the grand bouillon, extract or purée of roast meat, fowls and vegetables have been added, and this is known as a consommé. This is, of course, most albuminous.

In framing a diet, due weight must always be given to the composition of the particular soup indicated.

GENERAL SERVING OF FOOD FOR INVALIDS

If the patient is always objecting to the food, it is quite possible that the manner in which the food is served is responsible for the patient's fastidiousness.

Everything should be served as daintily as possible, the dishes, glasses, and traycloth being thoroughly clean. We must remember that to most invalids the meals are the events of the day, and too much care cannot be taken to ensure that everything regarding the food should be done in as attractive a way as possible. Patients should not as a rule be consulted as to their meals, but there should be an endeavour to discover beforehand what the patient likes. Any particular fancy as to sweetness or otherwise should be remembered. Untouched food should never be left in an invalid's room, but should be put aside in a cool place, and no food should be cooked or prepared in the invalid's presence if it can be done elsewhere. Food should never be tasted, or cooled by being blown upon, as these disgust the patient.

In the case of *helpless patients* who cannot feed themselves nourishment may be given by the spoon, or the feeding cup. When the patient is very helpless a useful device is to pull out the cheek, by inserting the finger between the gum and the cheek, and then introduce the fluid nourishment slowly at one side. A tea-spoon is the most convenient size of spoon for child feeding, and a dessert-spoon for the adult. As to drinking cups, the shape made with three handles is very convenient for the patient to use when feeding himself; but when the services of a nurse are required, a small boat-shaped feeding cup with a curved spout and about three inches of rubber tubing attached is the most useful. In the latter case great care must be taken to maintain thorough cleanliness of the apparatus in use.

In feeding with a spoon or feeding cup it is certainly easier for the patient to have the head raised if it is permissible; the nurse in these cases passes the left arm under the pillow on which the patient is lying, and gently raises the head.

Nasal feeding is called for in some cases of insensibility, melancholia, or other variety of insanity, and after tracheotomy. This method of supplying nourishment is very important. Half a pint to a pint of fluid food can be given at one time. The apparatus is simple; the small glass barrel of a urethral syringe has about 10 inches of narrowish rubber tubing attached. This is well oiled, and is passed gently along the floor of the nose in a backward and slightly outward direction; it slips over the posterior surface of the velum palati, and from thence into the pharynx and gullet. Patients soon become tolerant of this method. The nutriment is slowly poured from a jug in the glass receiver held directly over the patient's face.

Rectal Feeding.—Nutrient enemata are most valuable in certain circumstances, enabling the patient to hold out and to maintain his strength until the crisis that called for their use is over. Life may be maintained by rectal feeding alone for several weeks if all due precautions are taken with regard to it. As the large bowel possesses powers of absorption, but very slight if any power of digestion, the food given in this way must have the ingredients transformed into that state which is most easily absorbed. If this is not attended to, the nutrition will be defective, and a very troublesome irritative state of the bowel is induced which may become an insuperable obstacle to further successful treatment of the case.

The foods selected for rectal feeding must therefore be bland and easily absorbed. Predigested albumin and starch, and also fat in the form of an emulsion, are all absorbed in the rectum. Therefore peptonised beef tea, peptonised milk gruel, or maltine and peptonised milk, are all most useful forms of food. If peptonised food is not at hand two teaspoonfuls of liquor pancreaticus should be added to a 3 ounce enema of beef tea or of milk gruel shortly before its injection. Wine or spirit can also be added, and are to a certain extent absorbed. If the patient is suffering from pyrexia grape-sugar may be added to the enemata to supply the fuel food required.

A good enema for occasional use is—1 egg, 2 tablespoonfuls of milk, and a tablespoonful of brandy, well beaten up together.

The following special rules should be observed in the administration of a nutrient enema. (See also "Enemata," vol. iii.)

1. Two to two and a half ounces is the average size of an enema for an adult. More is apt to set up peristalsis.
2. The enema should be injected slowly and gently.
3. The temperature of the enema should be lukewarm, about 105° F.
4. The nozzle of the syringe should be lubricated with oil or vaseline, and care must be taken in the introduction of the syringe that the same spot of mucous membrane is not pressed upon each time. After injection there should be gentle pressure over the anus with a soft pad.
5. Care must be taken that the rectum does not become loaded with fæces. A preliminary washing out the bowel with tepid normal saline solution (75 per cent NaCl) at least once in twenty-four hours is very desirable.
6. A few drops (5 to 20 mins.) of tinct. opii given in every second enema is useful when the enema is retained with difficulty, and also as a prophylactic measure.
7. Not more than one enema every six hours should be given if possible.

Recourse may be had to the use of various forms of nutrient suppositories. Of these there are many excellent forms of prepared nutrient suppositories now in the market, which can be used to alternate with the home-made peptonised materials. It is advisable to bear in mind that the sediment from some of these preparations may possess rather more irritating properties than home-made preparations. Hence the great value of the occasional douching of the bowel above referred to.

Milk

A thorough understanding of the chief points to be attended to in the administration of milk is very important, as it is so often the chief and only agent we have to fall back upon in cases of illness.

Good milk should contain 3 to 4 per cent of fat. Its freshness and purity are essentials.

Milk should be obtained fresh twice daily, and should never be kept standing in the sick-room. It very rapidly becomes contaminated by bad air and emanations of decomposing substances, and thus soon loses its freshness. It should be brought into the sick-room in very small quantities, and should be kept covered. Unless the source of the milk is beyond suspicion the milk should be boiled or scalded.

Boiling most readily kills the germs, but unless a patient likes boiled milk it is almost impossible to get him to take it.

Boiled Milk.—If the milk is to keep some time it must be boiled when quite fresh. This is best carried out in a lined saucepan that has been rinsed out with water, or in a milk-can standing in a saucepan of boiling water.

The fire must be clear to avoid smoking. The milk must not be brought to the boil too slowly or it will curdle; and for the same reason it must only boil for a few minutes. As boiled milk that has been kept for several hours will not stand being boiled again, and hot boiled milk is more palatable than cold, therefore one has to fall back on sterilising or Pasteurisation of the milk to avoid waste. This consists in keeping the milk at a temperature of about 158° F. for twenty minutes; this is sufficient to kill most germs, but not the spores nor the lactic acid producing bacteria. This form of cooking does not alter the flavour of milk.

There are many sterilisers in the market, and the process is fully described under "Infant Feeding" (vol. v. p. 215); but it can be roughly done for occasional use by placing the milk in a deep jug and standing it in a pan of water, the water standing at a higher level than the milk. Boil the water for twenty minutes, but do not let the milk boil. This does not alter the flavour of the milk.

The amount of milk that should be taken per diem by a sick adult person in bed, when milk is the sole diet, is about four to five pints.

The possible disadvantages attendant on milk administration are, as a rule, easily overcome. Of these the more important are difficulties in connection with its curdling. In health, the normal curd usually readily redissolves in the alimentary canal; in illness, either from the want of digestive agents, or over-acidity of the stomach, a hard firm curd may form, which is an irritant to the whole intestinal surface, producing pain, flatulence, diarrhœa, or constipation, and may be discharged unchanged in the stools in some cases. This hard curd may be very dangerous in intestinal ulceration, as after typhoid. To avoid this the milk may be simply diluted to a third or a half by some mineral water, such as potash, Seltzer, soda, or an ærated plain water; or it may be diluted, and the formation of anything but a fine granular curd prevented, by mixing the milk with barley or toast water.

Clear Barley Water

2 oz. pearl barley (Robinson's).
1 pint boiling water.
Rind of half lemon.
Sugar to taste.

Method.—Wash barley well and put into a jug with thinly peeled rind of a lemon, and a little sugar. Pour freshly-boiled boiling water on barley. Cover jug and stand till cold. Strain, and it is ready for use.

Thick Barley Water

(Barley can be used twice.)

2 oz. pearl barley.
2 pints cold water.
Rind and juice half lemon.
Sugar to taste.

Method.—Wash the barley well, put it into a saucepan with cold water to cover it. Bring the water to the boil and then pour it away. Rinse the barley with cold water and rinse out the saucepan. (This is to blanch the barley, and if it were not done, the colour of the water would be dark.) Return the barley to the saucepan with two pints of cold water and the thinly peeled rind of half a lemon. Simmer slowly for two hours, strain, sweeten, and strain in lemon juice. This is served hot or cold. Time, two hours.

Note.—Omit the lemon juice if it is to be mixed with milk. It is a refreshing drink and a favourite with most invalids. A useful diluent for milk.

Toast Water

1 slice crust of bread.
1 pint cold water.

Method.—The crust of bread is better than slice, as it does not turn sour so soon.

Toast well on both sides until dry and nicely browned—not burnt. Have the fresh cold water in a jug, and break the toast into pieces and put into it. (If the water is poured on to the toast, it would be thick and not clear.)

Cover the jug, and let the toast remain soaking until the water is the colour of sherry wine. Strain and serve cold. This makes a refreshing drink with a little lemon juice added, and is also a useful diluent for milk, breaking up the curd.

If the patient is troubled with symptoms of acidity, an alkali such as carbonate of magnesia, as much as will lie on a sixpence, or a third part of lime-water may be added. This neutralises the acidity, and also prevents clotting.

Diarrhœa coming on while patient is on a milk diet is usually due to curd, and the above methods will usually stop it. The following can also be tried:—

Flavoured Milk

½ oz. ground rice.
½ pint milk.
Sugar.
A few pieces of cinnamon.

Mix the milk with the ground rice, pour into a small lined saucepan, allow it to simmer by the fire for half an hour. Do not let the milk boil over, and stir occasionally with a wooden spoon.

Sweeten to taste. Add some little bits of cinnamon stick, and serve hot or cold.

Rice, tapioca, and sago can be used in the same way. The flavouring can be varied with cloves or nutmeg.

If constipation is present it can generally be overcome by the adding ʒj of soda bicarbonate to the diluted milk, or by diluting some of daily allowance of milk with Vals or Vichy water.

Whey.—If milk is badly tolerated even after diluting, whey is often available, and can be made very palatable. It is a pleasant stimulant drink food; the milk sugar in it gives a certain food value, and this can be increased by the addition of one teaspoonful or more of lactose added to the pint. The absence of curd makes it easily digestible. Whey can be made in the following ways:—

Whey

1 pint new milk.
1 teaspoonful of rennet (Mackay's is tasteless).

Method.—Warm the milk in a basin to a lukewarm temperature (blood-heat, the temperature of new milk), and stir in the rennet.

Let it stand in a warm place about fifteen minutes, until the curd forms and the whey is quite clear. When cool, break up curd and strain off the whey through a piece of clean muslin.

Lemon Whey

½ pint milk.
1 tablespoonful lemon juice.
Sugar to taste.

Prepare as wine whey, using lemon juice instead of sherry wine. Time, five minutes.

Wine Whey

1 gill of milk.
½ gill sherry wine.
1 lump of sugar.

Rinse out saucepan, put in milk and sugar, and bring to the boil. When boiling, add the wine and the milk will curdle. Let it boil again and strain through a piece of muslin.

Cream Whey

This can be added to the ordinary whey or to the wine whey.

Add two tablespoonfuls of cream to half pint of whey.

CREAM is most useful in cases where feeding up is required, due to its large quantity of fat. It may be added to whey to increase the nutritive value of infants' food when attempting to make an artificial mother's-milk.

It is very largely used in invalid cookery and in the dietary of the aged to supply the place of oily fat food, and is added to soups, fish creams, blanc-manges, etc.

Again cream is of great use in the dietary of the diabetic, taking the place of milk and whey.

Skim milk is an excellent beverage in pyrexial states where fresh milk is too rich.

Butter Milk.—This is slightly more digestible than ordinary milk. The lactic acid present shows there must be a diminution of lactose, and this makes it valuable (vol. ii. p. 528) in the thirst of diabetes.

Koumiss—Fermented Mare's Milk.—This preparation has been used with a certain amount of success by consumptives in Russia. The secret of the cure is that the milk is slightly fermented, highly digestible, and large quantities can be taken without producing dyspepsia. The Russian mode of cure is to rise early and to take a glass of koumiss every half hour, with the exception of two hours preceding dinner and supper.

Kephir is a similar product prepared in the following manner from cow's milk :—

Kephir

3 gills new milk.
One small piece sugar.
½ gill boiling water.
German yeast size of a nut.

Quantity for each pint bottle.

Take the milk as new as possible. Fill the bottle nearly full of milk and water, and put into each a small piece of yeast and a lump of sugar.

Cork them tightly with new corks and fasten them down securely with wire. (The corks should be soaked for some time before being used.) Keep in a warm place and shake them two or three times a day for from four to six days.

This becomes very effervescent, and unless great care is used in opening a considerable quantity may be lost.

Koumiss and kephir are both more easily digested than ordinary milk, and owing to the destruction of a part of the sugar they are useful in various disorders of the digestive tract.

"Protene," the pure caseinogin from milk prepared as a flour, can now be obtained in the form of bread and biscuits (see vol. ii. p. 527). "Plasmon," a somewhat similar product, obtained from the proteids of milk in the form of a granulated soluble powder, is a recent useful addition to invalid dietetics. It can be added to gruels, meat teas, puddings, etc.

Milk sugar, or lactose, has the advantage of not being very sweet. It may be added to milk soups or other liquids to increase the food value without appreciably altering the flavour.

Eggs.—Next to milk an egg is one of the most valuable articles of an invalid's diet. Its composition is as follows:—

	Water.	Proteid.	Fat.	Ash.
White . . .	85·7	12·6	0·25	0·59
Yolk . . .	50·9	16·2	31·75	1·09

The ash is exceedingly rich in an organic iron compound and phosphorus, hence its acknowledged usefulness in the dietary of children in chlorosis.

The excellence of an egg depends upon it being perfectly fresh and properly cooked. If cooked too long it becomes indigestible, but a lightly cooked egg is as digestible as a raw one.

Raw eggs can be served in the following ways:—

Prairie Oyster

1 fresh egg.
1 tablespoonful of vinegar.

Put the vinegar into a small cup and break the egg into it. Serve at once. This is a very digestible way of serving an egg, and it is very cool and refreshing.

The next three recipes give the way of making up raw eggs into nourishing stimulant drinks. By adding nutmeg, cinnamon, or lemon juice the flavour can be altered.

Egg Drink

1 egg.
1 teaspoonful sugar.
1 tablespoonful sherry wine.
1 teacup of milk.

Beat up the egg, add to it the wine and sugar, and beat together with a fork slightly, and strain through a fine wire strainer.

Heat the milk in a small saucepan, and when almost boiling pour it on to the egg, stirring all the time. Serve hot.

This can be made without wine, flavouring with cinnamon or lemon juice. The yolk of the egg only may be used, and soda water instead of milk.

Plain Egg Flip

1 teacup of milk.
1 teaspoonful of sugar.
1 white of egg.

Boil the milk or make it thoroughly hot; beat up the white of egg to a stiff froth. Pour the boiling milk over the white of egg, stirring all the time. Add sugar to taste, and serve.

Rich Egg Flip

1 white of egg.
1 tablespoonful of cream.
1 tablespoonful brandy.
Sugar to taste.

Beat up the white of egg stiffly. Add to it the brandy and cream, with a little sugar if wished. Mix very thoroughly and serve.

Cooked eggs may be served by themselves in several ways.

Boiled Egg

Put enough water into a small saucepan to entirely cover the egg. Allow the water to boil; lower the egg gently into it. Draw the pan to the side of the fire, and allow the water to simmer slowly. Cook the egg for 3½ to 4 minutes, then lift out and serve at once. If it is allowed to stand it becomes hard.

Poached Egg

1 egg.	Lemon juice.
Boiling water.	Pinch of salt.

Round of toast.

Break the egg into a cup, keeping the yolk whole. Into a small saucepan of boiling water, add a pinch of salt and a squeeze of lemon juice. Draw the water to the side, and when just off the boil slip the egg carefully into it. Cook slowly for three minutes, when the white should be quite set. Lift it out with a small fish slice or perforated spoon, and trim off any ragged edges of white. Place on a square of newly-made toast and serve at once.

If required this egg can be made richer by being served on buttered toast, or a little hot cream poured over it. Also a poached egg served on two tablespoonfuls of carefully prepared spinach is a very nice dish. The method of preparing spinach, see p. 488.

Rumbled Egg

1 egg.	Pepper and salt.
1 tablespoonful milk.	¼ oz. butter.

A piece of toast.

Heat in a small saucepan the milk. Pour into the hot milk the egg beaten up in a cup with pepper and salt.

Stir quickly over the fire until it begins to thicken, remove it then from the fire and continue stirring until it forms a creamy mixture.

Put on a piece of newly-made toast and serve at once. If cooked too long, or allowed to stand, it becomes leathery and indigestible.

Baked Egg

Grease a china or paper ramequin case thoroughly; into this carefully break an egg. Add pepper and salt and a teaspoonful of cream.

Place in a moderate oven and bake three or four minutes until lightly set, and serve at once.

Eggs also form the bases of omelets, custards, soufflés, and are added to many farinaceous puddings to improve them.

PREPARED AND PREDIGESTED FOODS.

These are mainly of two kinds: (*a*) those in which there has been a predigestion of the carbohydrates with malt; (*b*) those in which the albumins have been converted into peptones and the carbohydrates into more soluble sugar, these changes being effected by the action of the active ferments of the pancreatic juice.

The so-called *prepared foods* are those sold by the manufacturers as suitable for babies and invalids. Their right to this description consists in their containing a certain quantity of malt which possesses the power of converting insoluble starch into glucose and grape sugar.

In all these foods the malt is mixed with some variety of baked flour The proportion of sugar and the presence or absence of milk (desiccated) are the varying factors in their composition. Those made with desiccated milk possess a higher nutritive value, and corresponding care must be taken in their administration when taken dissolved in milk. On the other hand, prepared foods in which milk is not an ingredient must be made up with milk. Those made with milk are decidedly more palatable than those mixed with water.

There are numerous brands of this class of food now in the market, many of which are distinctly valuable in practice. While it is impossible to enumerate all the varieties, reference may be made to Allen and Hanbury's, Benger's, Mellin's, and Savory and Moore's, as these are the brands which the writer can safely recommend from personal experience.

They are all taken mixed with fresh milk.

Of the Allenbury series the third food is the best suited for invalids. Nos. 1 and 2 are prepared with desiccated milk, and though very satisfactory for young infants, the taste of them for adults is not quite so palatable. No. 3 has only a certain proportion of its starch converted into a soluble carbohydrate; this leaves some starch (although cooked and rendered easy of digestion) for the digestive powers to act upon. This may with advantage be used as an intermediate food during convalescence when the digestive powers are slowly regaining their function. The great advantage of having a large choice of malted foods lies in the fact that the patient can get a certain change in the flavours and dishes even when on a very restricted diet.

By the use of malt, either in the form of a malt infusion or the extract of malt, many patients who experience much difficulty in digesting the starchy constituents of food, will find that they can do so quite comfortably.

The following valuable recipes may be mentioned :—

Malt Infusion (Sir W. Roberts' recipe)

Crushed malt, 3 oz.
Cold water, ½ pint.

Mix the malt thoroughly with the cold water in a jug. Let the mixture stand nine hours. Decant off the liquid carefully, and strain this through muslin until it comes out clear and bright.

This has a colour like dark sherry and a faint maltish taste.

It should be freshly made as it is liable to ferment.

Malted Gruel

The gruel may be made of any of the farinaceous substances—oatmeal, Quaker oats, farina, arrowroot, barley, or lentil flour, preferably those from prepared flours. For method of making gruel, see p. 477.

When cool enough to swallow add the malt infusion or an extract of malt. About one tablespoonful of the infusion or a teaspoonful of the extract is sufficient to digest a plateful of gruel. The action is very rapid, and in a few moments the gruel becomes thin from the transformation of the starch into maltose.

Several such dishes can be made out of invalid food that form a pleasing variety. Any invalid food can be used as a basis for these recipes. Benger's food is especially well adapted for it.

Invalid Food

1 oz. of "invalid food."
4 tablespoonfuls of cold milk.
½ pint boiling milk.

Mix the food into a paste with four tablespoonfuls of cold milk. Add this gradually to the half pint of milk, which should be almost boiling; keep stirring all the time.

Set aside in a warm place for about twenty minutes. Then boil the whole for a minute, stirring all the time, and strain if necessary.

This can now be taken as an invalid food or used in the making of various dishes, and the recipes are those given by Benger.

Custard Pudding

½ pint of the "invalid food" made with milk.
2 eggs.
2 teaspoonfuls of sugar.

Prepare the food as in the given direction on the tin. After it has been allowed to cool add to it the sugar and the eggs well beaten.

Pour into a small greased pie-dish, and bake in a moderate oven from 10 to 15 minutes. Flavouring, such as nutmeg, cinnamon, or grated rind, may be added. This may be served hot or cold.

Batter Pudding

½ pint of the "invalid food" made with milk.
2 eggs.
1 pinch of salt.

Take the prepared food and give it a final boiling. Let it cool slightly, and add the salt and eggs well beaten.

Pour the mixture into a well-greased basin, cover with greased paper, and strain slowly for half an hour. Do not fill the basin too full as the pudding will rise. When ready it will be firm to the touch.

It can be served with sugar, preserves, or meat gravy.

Blanc-mange

½ pint of "invalid food" made with milk.
3 teaspoonfuls of corn flour.
2 teaspoonfuls sugar.
Flavouring, vanilla or lemon.
2 tablespoonfuls of cream.

Mix the corn flour and sugar in a basin and gradually add the food, mixing well together. Pour into a saucepan and stir over the fire until boiling. Boil for five minutes. Add the flavouring and cream.

Pour into a wetted mould, and put into a cool place until set. When ready turn out and serve with stewed fruit.

Invalid Food Jelly

½ pint of "invalid food" made with milk.
½ oz. French leaf gelatine.
2 teaspoonfuls of castor sugar.

Make the invalid food according to directions. Add while hot the gelatine, and stir over the fire until dissolved.

Add the sugar and any suitable flavouring, such as port or sherry wine.

Pour into a wetted mould until set.

Turn out and serve cold.

PEPTONISED FOODS.

Peptonised foods should only be employed when it is necessary to assist the digestive organs for a time, by giving the alimentary canal some degree of physiological rest. As soon as the digestive organs have regained their vitality their use should be abandoned. Dr. Fothergill tells the story of the Nottinghamshire farmer whose son had been largely fed on peptonised food, who said, "It is just making a pauper of his stomach, doctor," and as a ratepayer he knew that paupers would not work if they could help it.

Pepsin and dilute HCl may be used to predigest proteid food, but it is more convenient to employ liquor pancreaticus, as it has less effect on the flavour of the food.

In preparing food by means of pancreatic ferments there are many reliable preparations that can be used. Liquor pancreaticus (Benger's) and Fairchild's zymine peptonising tubes may be mentioned. It is well to remember that the ferments must not be added if the temperature of the food is higher than 140° F. Roughly speaking, this is a temperature that will not scald the mouth. If a greater heat is applied the ferment is destroyed.

The extreme solubility of predigestive products, whether starch or albumin, makes them appear thin and watery, but to a delicate invalid without an appetite this is an improvement, as the sensation of thickness is generally much objected to.

If peptonised foods are prepared with care there is no reason why these foods should have an unpleasant taste and smell. The following methods of preparation of different predigested foods may be commended:—

Peptonised Milk

Milk, 1 pint.
Water, ¼ pint.
Liquor pancreaticus, 2 teaspoonfuls.
Bicarbonate of soda, 20 grains (½ small teaspoonful).

After allowing the milk to stand skim the milk. Dilute the skimmed milk with ¼ pint of water, heat to a temperature of 140° F.

Mix with the warm milk 2 teaspoonfuls of liquor pancreaticus and 20 grains of bicarbonate of soda.

The mixture is poured into a covered jar, with cosy over it, and put near the fire. Let this stand for 1½ hours.

Bring the mixture to the boil for two to three minutes to arrest any further fermentative action, which makes the milk unpalatable. Now add the cream that was removed, and it is ready for use.

Cold Process.—Mix the peptonising agent in cold water and cold milk as usual, and immediately place the bottle on ice, without subjecting it to any heat.

When needed pour out the required portion, and use in the same manner as ordinary milk.

It is recommended to try the milk prepared by the cold process in those cases in which food is not quickly digested after ingestion, and in which the digestive functions are impaired, or even practically suspended. It has been found in many such cases that the peptonising principle exerts sufficient action upon the milk in the stomach to insure its digestion and proper assimilation. If the milk so prepared be not well borne, or any evidence appear of its imperfect digestion, it should be sufficiently predigested—peptonised—by the usual warm process.

Milk by the "cold process" is especially suited for dyspeptics and persons who ordinarily find milk indigestible. This milk has no taste or evidence of the presence of the peptonising agent.

Effervescent Peptonised Milk.—Put some finely cracked ice in a glass, and then half fill it with cold Apollinaris, Vichy, or carbonic water as preferred, then quickly pour in the peptonised milk and drink during effervescence.

Peptonised milk may be made agreeable to many patients by serving with a little grated nutmeg, sweetened or flavoured with a little brandy, etc.

Peptonised Gruel.—The gruel may be made from any farinaceous article in common use—oatmeal, Quaker oats, farina, arrowroot, barley, pea or lentil flour.

The gruel should be well boiled. It is placed in a covered jar when it is below the temperature of 140° F. Add liquor pancreaticus in proportion of 1 tablespoonful to 1 pint of gruel; and the jug is covered and kept warm as before. After standing for two hours boil the mixture for five minutes and then carefully strain.

The pancreatic ferments have two actions on the farinaceous matters:—

1. The starch of the meal is transformed into sugar.
2. The albuminoid matters are peptonised.

Peptonised Milk Gruel.—This was very highly spoken of by Sir W. Roberts. It is an artificially digested milk, and forms a complete and highly nutritious food for weak digestions.

A thick gruel is made from any farinaceous material, see p. 477.

To the boiling gruel add equal quantity of cold milk. This will then have a temperature of about 140° F.

To each pint of gruel and milk add 3 teaspoonfuls of liquor pancreaticus and 20 grs. of sod. bicarb.

Let it stand covered in a warm place for two hours, boil for five minutes, and then strain.

The bitterness of the predigestive milk is almost completely covered in this peptonised milk gruel, and most invalids will take it without the least objection.

Peptonised Milk Lemonade

Juice of 1 lemon.	1 gill peptonised milk.
1 tablespoonful of sugar.	1 gill effervescing water.

Some small pieces of ice.

Fill a small tumbler one-third full of cracked ice. Add the water, lemon juice, and the sugar to the ice, and then fill up the glass with milk.

Peptonised Milk Punch.—Take a small tumbler, and fill it about one-third full of finely crushed ice, pour on to this a tablespoonful of St. Croix rum and half a tablespoonful of brandy, then fill the glass with peptonised milk, stir it well, sweeten to taste, and sprinkle over the top of the mixture a little grated nutmeg.

Peptonised meat broths and teas can be made in the following manner:—

½ lb. lean beef finely minced.
1 pint water.
20 grains bicarbonate of soda.
1 tablespoonful of liquor pancreaticus.

Mix together and simmer slowly for 1½ hours; when it has cooled down to 140° F. add liquor pancreaticus. Keep it warm under a cosy for two hours; occasionally shake it up. Then decant off the liquid portion and boil for five minutes. This has a relative value in nitrogenous materials almost equivalent to milk. The beef tea can be seasoned with celery seed cooked in the tea, or vegetable flavourings can be added by placing a small muslin bag containing some carrot, turnips, parsley, onion, in the tea while cooking, and afterwards removing it. If desirable the tea can be thickened by the addition of a roux (paste) made with a little baked flour (p. 474.)

In addition to home-made peptonised beef teas and juices there are a large number of reliable proprietary medicines, of which may be mentioned Armour's nutrient wine of beef peptone, Benger's peptonised beef and chicken jelly, Brand's beef, mutton, veal, and chicken peptone, and Fairchild's panopeton. This latter preparation claims to present bread and meat in a perfectly soluble form; it is certainly very palatable.

Messrs. Savory and Moore have manufactured for the use of invalids and convalescents a very palatable form of peptonised cocoa and milk, and also a concentrated peptonised milk and coffee (café zylak). These are delicious preparations and can be much enjoyed even by people who cannot take cocoa or coffee in any other form.

Recently a new preparation called somatose has been placed on the market. It claims to be a very concentrated and partially digested proteid substance, the albumin being mostly converted into albumose. It is a yellowish powder freely soluble in water, and forms almost a tasteless solution. It is useful in increasing the nutritive value of beef tea, etc. to which it may be added.

Peptonised Soups, Blanc-manges, and Jellies.—With a little ingenuity a variety of peptonised dishes can be obtained by preparing soups, blanc-manges, and jellies with peptonised elements. There is no difficulty in doing this if it is always remembered that if any peptonised fluid is used it must be boiled in order that the action of the ferment may be arrested.

For soups, use peptonised gruel, p. 471, instead of water. For the making of blanc-manges (p. 479); peptonised milk is added to the cream. For jellies mix the liquor pancreaticus with gelatine, the flavouring matter to be employed in the proportion of 2 teaspoonfuls to the pint; allow it to stand for 1½ hours in a warm place, and then check the further action by boiling. (For jellies see p. 480).

Peptonised Oysters

Take half a dozen large oysters with their juice and half a pint of water. Heat in a saucepan until they have boiled briskly for a few minutes. Pour off the broth and set aside.

Mince the oysters finely, and reduce them to a paste with a potato masher in a wooden bowl.

Now put the oysters in a glass jar with the broth that has been set aside, and add—

Liq. pancreaticus, 2 teaspoonfuls.
Soda bicarb., 15 grains.

Let the jar stand in hot water or a warm place where the temperature is not above 115° for 1½ hours.

Then pour into a saucepan and add half a pint of milk.

Heat over the fire slowly to boiling-point.

Flavour with salt and pepper or condiments to taste, and serve hot.

There will be found a few very small pieces of the oysters undigested, and these may be strained out or rejected in eating the soup, but will not be unacceptable to the stomach, except in very rare cases.

The *milk* will be sufficiently digested during the few minutes which will elapse before the mixture boils, *if heated gradually.*

Be sure to boil the peptonised oysters to finish the process.

FOOD IN PYREXIAL STATES

When fever is present two important indications are the supply of *fluid* in the form of pleasant beverages to overcome the dry and parched sensations in throat and fauces brought on by the high temperature; and, secondly, the administration of a liberal supply of carbohydrates, which is necessary to meet the increased oxidation in the tissues resulting from the high temperature.

With suitable feeding the wasting is less, and the patient does not become so reduced, and, consequently, the convalescence is more rapid.

Beverages.—For the thirst the following recipes may be given for refreshing drinks, but they all have a very low nutritive value. If made with barley (p. 463) or rice water, instead of plain water, the value is slightly increased. Again, the addition of one teaspoonful of milk sugar or lactose to the pint increases the value considerably without affecting the flavour.

Rice Water.—Take 2 ounces of rice into an enamelled saucepan with 3 pints of water, and boil for 2½ hours. Stir it frequently and skim carefully.

Strain into a jug through a fine wire sieve, and rub through the glutinous part, but not the hard portion.

Put in no flavouring unless ordered by the practitioner.

Apple Water

1 lb. of apples.
½ lb. brown sugar.
1 gallon boiling water.

Cut up the apples into quarters; take them and put into a jug with the brown sugar, and pour the boiling water over it. Let it stand until cold; pulp the apples and the fluid through the colander. Bottle for use. Do not cork the bottle. Keep in a cool place.

Apple Toast and Water.—Toast a piece of bread slowly until it gets quite black. Placed in a jug of apple water for three-quarters of an hour, then strained, makes a very nice and refreshing drink.

To make Apple Barley and Apple Rice Water.—Boil ½ lb. of rice or ¼ lb. of barley, blend with the apple water, and then strain it.

In spring and summer rhubarb, green gooseberries, black and red currants, and raspberries, may all be made into nice cooling drinks if used in the same proportion.

Imperial Drink

½ oz. cream of tartar.
Juice of 1 lemon.
2 tablespoonfuls of sifted sugar.

Place the ingredients in a jug, and pour over a quart of boiling water, and cover until cold.

Lemonade

(*a*)
Juice of 1 lemon.
1 pint of water.
1 ounce of sugar.
1 egg.

Dissolve the sugar in the water; add the juice of the lemon; beat up the white of egg, and add this.

This makes a very palatable drink with slight nutritive value.

(*b*)
4 lemons.
¼ lb. of loaf sugar.
3 pints boiling water.

Rub some sugar on the rinds of two of the lemons until it is yellow.

Strain the juice of the four lemons; put the sugar and juice into a jug, and pour over the water. Cover it until it is cold.

Orangeade

2 oranges.
Juice of 1 lemon.
2 or 3 lumps of sugar.
1 pint boiling water.

Wipe the oranges with a damp cloth, and peel the rim off one of them very thinly. Put this into a jug with sugar, and strain in the juice from the oranges and lemons. Pour on the freshly boiled water. Cover closely until cold, and then strain.

Black Currant Drink

2 tablespoonfuls black currant jam.
1 teaspoonful arrowroot.

Take the black currant jam and boil it in a quart of water. Cover it and stew gently for half an hour; then strain it, and set the liquor again on the fire. Mix the arrowroot in cold water; pour over it the boiling liquor, and keep stirring. Then let it get quite cold.

(This is a very pleasant drink, and specially nice for a rough throat.)

Malt extracts (see p. 468), with effervescing water, are also excellent as beverages. They quench the thirst, and supply grape sugar (maltose), soluble dextrine, and a small quantity of soluble albuminoids.

MEAT BROTHS AND EXTRACTS OF TEAS.—Meat infusions of beef, mutton, veal, and chicken are now in considerable demand. These are to be regarded more in the nature of pleasant, palatable, and stimulating beverages than as foods. They may be taken hot, cold, or iced. To this class belong the large class of meat extracts now in the market—Bovril, Liebig, Vigoral, Vimbos, and mutton extracts, *e.g.* Hipi.

Home-made Beef Teas.[1]—In their preparation it is well to employ beef from the buttock, rump, or thick flank, in preference to shin of beef. In the process of cooking the tea must not be allowed to boil, otherwise the juices are not extracted owing to the insoluble albuminous coating formed on the surface of the meat by the boiling process. In every case care must be taken to ensure that all the fat is removed from the surface before serving.

There are various ways in which the beef tea may be thickened, *e.g.* with egg, tapioca, bread crumbs, or baked flour, thus adding much to the nutritive value.

The baked flour is prepared as follows:—

Baked Flour.—Spread some white flour thinly on a dish, bake it in a moderate oven for about an hour until it is a delicate cream colour. Take it out and place on a sheet of kitchen paper, when cool roll it smooth with a rolling pin, pass it through a wire sieve, and then keep in a canister or bottle. This may be used for thickening meat teas and broths.

Puddings made with this form of flour are more digestible.

In serving soup to an invalid do not fill the cup or basin too full. A thin slice of old bread, or a piece of freshly toasted bread, or an unsweetened rusk should be served with the soup.

The following recipes may be commended.

A glance at the composition of each will convey some idea of their relative value as nutritive fluids; some of them are mere stimulants.

Very little vegetable flavouring is given in the accompanying recipes, but if permissible celery seed, or a muslin bag containing a variety of vegetables, may be cooked with the beef tea and afterwards removed.

Beef Tea

1 lb. lean juicy beef.
1 pint cold water.
1 small teaspoonful of salt.

Method 1. Wipe the meat with a damp cloth and then place on a board, and with a sharp knife shred down as finely as possible. Keep back any pieces of connective tissue and fat.

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[1] Should always be made from freshly killed meat.

Place in a basin with salt and water, stir it well up, and cover the basin with a plate, and if time permits let it stand for half an hour. Then pour the contents of the basin into a clean lined saucepan, place the pan over a slow fire, and whisk it well until it almost reaches boiling-point; *do not let it boil.* Then draw the pan to the side of the fire, put on the lid, and allow the beef tea to simmer as slowly as possible for about 10 to 15 minutes. Strain through a coarse strainer, pressing the meat as dry as possible with a wooden spoon. Remove all the fat before serving.

In cases where it is desirable to obtain the maximum of extractives in the tea, *i.e.* to form a very stimulating beverage,—

Method 2. Use the same proportions of meat and water, and prepare it in the same way as above, but afterwards place the meat in a beef-tea jar, and cook slowly in the oven for four hours. The jars are strong stone jars with a strong screw lid, and can be obtained from Dall and Company, Edinburgh. They are made in various sizes.

Caution.—After making beef tea in this way the jar must not be opened until the contents are cold. If opened before it is cold the steam rushes up and is very apt to scald the cook's face and arms.

If preferred, chicken, mutton and veal, or veal tea can be made in a similar manner. In making chicken tea, the legs and dark parts of the fowl can be used for soup while the white portions can be served in some other way. The fillet of veal is the best portion to order. In the mutton and veal tea, equal parts of the two meats are used; this gives a very delicate flavour, and is much appreciated by many invalids.

Thick Beef Tea

½ pint of made beef tea.
1 yolk of egg.
1 teaspoonful Grôult's tapioca.

Warm the beef tea and sprinkle in the tapioca, stirring all the time.

Let it simmer slowly by the side of the fire for about ten minutes until the tapioca turns quite clear.

Beat up the yolk of an egg in a basin or cup with a fork, pour the beef tea gradually over it, stirring all the time, and it is ready for serving.

½ pint of made beef tea.
1 teaspoonful of arrowroot.
1 tablespoonful cold water.

Mix the arrowroot and water in a small basin until quite smooth. Then add it to beef tea that is being warmed in a pan, stir well for a few minutes to prevent it becoming lumpy. Then simmer slowly for ten minutes.

Beef-Tea Gruel

1 tablespoonful fine oatmeal.
1 gill cold water.
1 gill beef tea.
Salt.

Make the gruel with oatmeal and water or milk as above.

When cooked add to it the beef tea, stir until quite hot, but do not boil.

Season to taste and serve hot.

Beef Purée

½ pint of good beef tea.
¼ lb. lean juicy meat.

Warm in a pan the beef tea that is already made.

Wipe and shred very finely ¼ lb. of meat, pound it well, and rub it through a fine wire sieve. Take a cup and make it thoroughly hot, into the cup put the pounded meat, pour the hot beef tea on to it, stir it well up, and serve at once.

Beef Essence.—One pound of meat will make one gill of essence.

Prepare meat as in last recipe shredding it down very finely.

Place in a jar with a pinch of salt, cover with a strong piece of greased kitchen paper.

Place the jar in a saucepan containing sufficient cold water to reach fully half way up the jar, and let it steam slowly for four to five hours.

When ready, strain through a fine strainer and press the meat well with the back of a wooden spoon to extract all the juice. This liquid is thus pure beef juice.

When cold this is in the form of a jelly.

Meat Juices.—This variety of food differs greatly in nutritive value from those extracts previously mentioned. The meat juice is extracted without any heat, and thus a large portion of the albumin is present. Brand's, Valentine's, and Wyeth's meat juices are all excellent preparations, and are to most people very palatable.

Raw meat juice can be prepared in the following manner; its great drawback is its decidedly objectionable appearance, which can be partially overcome by serving in a red glass or a cup.

¼ lb. best rump steak.
½ pint cold water.
Pinch of salt or sugar to taste.

Prepare the meat in the same way as for Beef Purée. Place the shredded meat in a basin with water and the salt. Stir it well with a fork, cover with a plate, and let it stand ¾ hour, stirring occasionally. The liquid will then be a bright red colour. Strain through a fine strainer, pressing the meat with the back of a wooden spoon.

This should only be made in very small quantities, as it very soon becomes rancid.

A recent patent preparation prepared by Brand, and termed "Fever Food," is most nutritive; it consists of essence of beef, eggs, cream. In appearance it resembles custard, and has a very agreeable flavour which is quite distinct from the meaty flavours of the beef juices and extracts.

MILK is the staple food in febrile state. If badly borne it may be taken as whey or diluted with Seltzer, soda, or potash water. The fat present in the milk is most useful, and this method of administration of fat is practically the only one available, as such patients have usually a marked intolerance of other forms of fat. In cases with intestinal weakness, *e.g.* typhoid fever and the like, the milk must be prevented forming too hard and tough a curd, and the measures suggested to remedy this should be carried out (page 462).

DIET DURING CONVALESCENCE

As the digestive tract is more or less involved in all cases of acute illness, convalescents may all be regarded as dyspeptics for the time being. Thus at first great care must be taken both in quantity and the quality of the food.

However, the digestive capacity of convalescents varies. There is, for instance, the healthy young adult recovering from some sharp attack of an infectious fever, in whom the digestive organs are only weakened, not organically affected. His convalescence is rapid; he is soon capable of eating and digesting everything, and in this class of case there is no necessity to try and tempt the appetite. The food can be simply prepared, the danger is that the patient will overeat. On the other hand, we may have a delicate child recovering from scarlatina complicated by nephritis. Here it would be absolutely wrong if we did not limit the quantity, and give very strict injunctions as to the quality of the food.

After typhoid fever, when convalescence is thoroughly established, any mass of undigested food, *e.g.* potatoes, or a piece of unmasticated meat, may be the cause of a relapse by setting up irritation in some recently ulcerated patch.

At first, in all cases, a continuance of the fever diet, of milk and potash, beef tea or meat infusions, and acid drinks, is indicated. This diet can be gradually extended to include easily digestible starches, either in the form of some prepared invalid food (p. 468), or as a home-made *gruel*, to which has been added malt infusion.

The two following recipes are useful for gruels:—

Oatmeal Gruel

1 tablespoonful fine oatmeal.
½ pint cold water.
Salt or sugar.

Put the oatmeal into a clean basin, and pour the water over it. Cover the basin and let it stand for at least half an hour, stirring occasionally. Then strain the liquid off into a small lined saucepan, and press the oatmeal as dry as possible. Stir over the fire until boiling, and let it boil from five to seven minutes. The thickness of the gruel is very much a matter of taste, if too thick add more water, or if too thin use more oatmeal.

Season with salt and sugar; wine or brandy may be added if required.

Milk Gruel

1 tablespoonful fine oatmeal.
½ pint milk.
Salt or sugar.

Made in the same way as last recipe, only milk is used instead of water.

Barley Meal Gruel

1 dessertspoonful barley meal.
½ pint milk.
A small piece of butter.
Sugar or salt.

Mix the milk very gradually with the meal, stirring until quite smooth. Take a small lined saucepan, rinse out with cold water, and pour the barley and milk into it.

Stir constantly over the fire until boiling, and let it boil about six minutes; season to taste, and serve very hot. This is better eaten with a little cream. The thickness can be regulated as in oatmeal gruel. This can be made into a very good drink by making a thick gruel with water, then thinning it down with port wine; heat thoroughly, but do not boil again.

As the conditions of the digestive tract improves the desire for food increases, and this must, to begin with, be met by giving *milk puddings*, and *eggs* in the form of drinks, *e.g.* egg flip, etc. or as baked *custard*.

In the following recipes for baked milk puddings, the eggs can be omitted if desired. The presence of eggs in a pudding greatly improves its flavour and appearance, but they make it richer and more difficult to digest. If the pudding is to be made with eggs it is always lighter when the white of the egg is beaten up separately, as this introduces air into the pudding and causes it to rise.

Care must be taken to thoroughly cook all the farinaceous and starchy elements of the food. For flavouring the most suitable are fresh lemon rind or juice, grated nutmeg, ground cinnamon, and essence of vanilla.

Water Arrowroot or Corn Flour

½ oz. arrowroot or corn flour.
½ pint cold water.
1 teaspoonful of sugar.

Put the arrowroot into a small basin; add to it a tablespoonful of cold water; break it with a wooden spoon until quite smooth.

Then pour on the rest of the water, mix well, and pour into a small lined saucepan. Stir this over the fire until it boils and thickens, then let it boil for 10 minutes to thoroughly cook the arrowroot.

Sweeten to taste and serve in a cup or small basin.

A little nutmeg may be grated on the top, and wine or cream added if wished.

Milk Arrowroot or Corn Flour

½ oz. arrowroot or corn flour.
½ pint milk.
½ teaspoonful of sugar.

Make in the same way as above, using milk instead of water.

Arrowroot or Corn Flour Pudding

½ oz. arrowroot.
½ pint milk.
1 egg.
1 teaspoonful of sugar.

Make in the same way as arrowroot and milk, and thoroughly boil the mixture for five minutes. Remove the pan from the fire; add the sugar and any flavouring.

Separate the yolk from the white; add the yolk to the arrowroot whenever it has slightly cooled; add a pinch of salt to the white, and beat it up to a stiff froth. Stir this lightly into the mixture and pour into a greased pie dish, wiping round the edges of the dish. Bake in a moderate oven until well risen and of a nice light brown colour.

Sprinkle some white sugar over it and serve at once, as it soon falls if allowed to stand.

Semolina or Ground Rice Pudding

¾ oz. semolina or ground rice.
¼ pint milk.
1 egg.
1 teaspoonful of sugar.

Put the grain into a small lined saucepan and the milk with it. Stir these over the fire with a wooden spoon until boiling, and boil for a few minutes until the semolina swells and thickens. Remove the pan from the fire; add the sugar and seasoning, and when it has cooled mix the yolk of the egg well into it.

Beat up the white on a plate with a knife to a stiff froth and mix thoroughly; pour the mixture into a pie dish and bake for 10 minutes; sprinkle with sugar and serve at once.

Rice Pudding

1 oz. whole rice.
½ pint milk.
1 teaspoonful of sugar.
1 egg.
Flavouring if wished.

Well wash the rice in several waters; put it into a lined saucepan with cold water to cover it; bring it to the boil and pour the water off. This helps to burst the rice more quickly.

Then pour in the milk and let the rice simmer by the fire until thoroughly cooked, then add the yolk of the egg and the white as described in the last recipe, and bake in a well-greased pie-dish.

Tapioca and Sago Pudding

¾ oz. tapioca or sago.
½ pint cold milk.
1 egg.
1 teaspoonful of sugar.
Flavouring.

If small crushed tapioca or small sago make as in semolina pudding.

If not, follow the directions—

Let the tapioca soak about an hour in a basin covered with milk.

Rinse out a small lined saucepan, turn the tapioca and milk into it, and stir over the fire until it comes to the boil. Then simmer slowly until it turns clear, stirring every now and then for about 20 to 30 minutes.

If it becomes too thick while cooking add a little more milk. Then finish off the pudding with eggs, etc. as in semolina.

A nice light pudding can be made by cooking the sago or tapioca with water instead of milk, and adding stewed fruit or claret or port wine to it.

Baked Bread-and-Butter Pudding

1 or 2 slices of thin bread and butter.
1 egg.
½ pint of milk.
1 teaspoonful of sugar.
A little grated nutmeg.

Cut some bread and butter rather thin, remove the crusts, and cut it into pieces about an inch square. Lay these into a small greased pie-dish, making the dish just about half full; beat up the egg in a small basin, and add to it nutmeg, sugar, and milk. Mix well together and pour over the bread in the pie-dish.

Allow the pudding to stand about 10 minutes until the bread gets thoroughly saturated, then bake in a moderate oven from 10 to 15 minutes until nicely browned on the top. Sprinkle with sugar.

Blanc-mange

1 gill of milk.
1 gill of cream.
¼ oz. isinglass.
Rind ½ lemon.
1 oz. sugar.

Rinse out a small saucepan and put into it the milk, isinglass, and thinly peeled rind of half a lemon. Let this stand by the side of the fire until the isinglass is dissolved and the milk well flavoured with the lemon. Stir occasionally, but do not let it boil.

Add the sugar, and strain into a basin to keep back the lemon rind. Add the cream, and stir occasionally until nearly cold. If not stirred the milk and cream will separate.

Pour into a small basin or mould that has been rinsed out with cold water, and put aside in a cool place to set. Turn out and serve with a little red or black currant jelly.

Baked Custard

2 yolks and 1 white of egg.
1½ gill of milk.
1 teaspoonful of sugar.
Flavouring.

Beat up the eggs in a basin with the sugar and flavouring; pour in the milk and mix again; strain the custard into a greased pie-dish. Stand the pie-dish in a Yorkshire pudding tin with some cold water round it.

Bake in a moderate oven from 15 to 20 minutes until firm and nicely browned.

A thin slice of sponge cake may be placed on the top of the custard before baking.

JELLIES

Calf's-foot jelly is very troublesome to make and to clear, and is no better than jelly made from gelatine.

Wine Jelly

1¼ oz. French sheet gelatine.
3 gills cold water.
½ gill lemon juice.
½ gill sherry wine.
1 tablespoonful brandy.
3 oz. loaf sugar.
Rind of 1 lemon, *cut*.
2 or 3 cloves.
1 inch of cinnamon stick.
White and shell of 1 egg.

(The proportion of gelatine is 2½ oz. to 1 quart.)

Put all the ingredients into a lined saucepan; whisk until they boil; remove to the side of the fire when the scum begins to rise to the top. Cover the top of the pan with a plate, and allow it to stand fifteen minutes. Strain through a hot jelly cloth, run through three or four times till clear, and when cold mould in a scalded wet mould.

Whipped Jelly.—Melt the jelly by standing the basin containing it in a saucepan of hot water. When dissolved, put into a large basin and with a whisk whip it until it is quite cold and should become a firm froth and perfectly cold. By varying the ingredients in the wine jelly a good many different forms can be obtained, *e.g.*

Port Wine Jelly.—Take 3 gills port wine, 1 gill water, and 1 tablespoonful of red currant jelly, instead of the sherry wine, lemon juice, and the large amount of water.

Orange and Lemon Jellies are made by substituting orange or lemon juice for the wine.

SUGGESTED DIETARY FOR A CONVALESCENT

As it is important for the practitioner to be ever ready with suggestions as to what individual patients may partake of, the following is a suggested dietary for five days. Doubtless in many cases a more rapid return to ordinary diet could be made, while in others the changes would require to be more gradually effected. The patient is supposed to have reached the stage when he is able to digest milk puddings when taken in moderation.

For bread stuffs—thin slices of toast, the crusty portion of a Vienna roll, sweetened and unsweetened rusks, toasted sponge finger biscuits, rice biscuits, and plain biscuits may be recommended.

As the patient advances, eggs, fish, sweetbreads, chicken, small birds, rabbit, game, follow one another. Then come mince, beef quenelles, eye of a tender mutton chop, a tender slice from a joint or steak. The patient should always be reminded of the necessity of eating slowly and masticating well. Vegetables must be cautiously allowed at first.

All meals should be very simple.

1*st Day*

7 A.M.—*If awake early.*—A small cup of freshly-made tea with cream, and small half slice toast.
8.30 „ *Breakfast.*—Saucerful of oatmeal gruel with cream, and a little piece of Vienna roll—crusty part.
11.30 „ A teacupful of beef tea unthickened, either hot or cold.
1 P.M.—Steamed whiting; half slice of bread.
Semolina or ground rice pudding.
4 „ Potash and milk with rice biscuit or sponge cake.
A little fruit.
6.30 „ Benger's food in any form (see p. 469).
9 „ Some variety of meat tea or infusion.

2*nd Day*

8 A.M.—Saucerful of hominy porridge or barley meal gruel, etc.
A small cup of tea, toast, and a very little butter.
11.30 „ A teacup of chicken tea or a little fruit.
1.30 P.M.—Baked or steamed fish, p. 482.
Spinach.
Blanc-mange.
4 „ Potash and milk, or a small cup of fresh tea, bread and butter, and sponge cake.
6.30 „ Invalid food, or peptonised cocoa and milk.
9 „ Some variety of meat infusion.

3*rd Day*

8 A.M.—A lightly boiled egg.
A small cup of tea.
11.30 „ A teacup of soup, or a little fruit and a drink of milk and potash.
1.30 P.M.—Rabbit. Chicken, roast—2 slices from breast. Pigeon, served with bread sauce.
A small helping of vegetable (not potato).
(Stewed vegetable marrow.)
Pudding. Apples cooked and water sago, eaten with cream.
4 „ A small cup of tea, bread and butter, and biscuit.
6.30 „ Invalid food.
9 „ Some variety of soup.

4*th Day*

8 A.M.—A piece of boiled or steamed haddock, whiting, or sole.
Tea, bread or toast and butter.
11 „ Soup or egg drink.
1.30 P.M.—Sweetbread or tripe.
Vegetable. Stewed tomato (do not eat skins).
Stewed fruit with custard.
4 „ Afternoon tea (avoiding rich cakes and pastry).
6 „ Poached egg on toast or spinach.
Milk and potash.
9 „ Cup of soup.

5*th Day*

8 A.M.—A few rolls of well-fried streaky bacon.
Toast and tea.
11.30 „ Egg flip, or soup, or milk and potash.
Fruit.
1.30 P.M.—A small slice of tender roast mutton, or the eye of a tender mutton chop.
Vegetable.
Jelly.
4.30 „ Afternoon tea (avoiding rich cakes and pastry).
6.30 „ Fish.
Milk pudding.
9 „ Cup of soup.

Fish.—Whiting, haddock, sole, are the most easily digested fish, and as such should come first in the dietary. They can be steamed and served without any sauce, or stewed in a little milk and served with a white sauce. Nothing is nicer for an invalid than a really well-fried fillet of sole or small whiting, with a slice of lemon to squeeze over it.

Steamed Fish.—This is the lightest and simplest mode of cooking fish for an invalid.

1 filleted haddock, whiting, or sole.
A small piece of butter.
A pinch of salt and white pepper.
A squeeze of lemon juice.

Cut the fillets of fish into neat-sized pieces; grease a soup plate or muffin dish with a little butter, and place the pieces of fish on this.

Sprinkle with a little salt and white pepper if it is allowed, and squeeze over some lemon juice, which helps to keep the fish firm and white.

Cover the fish with a piece of greased white paper and then with a lid or basin. Place this over a pan half full of boiling water, seeing that the plate fits well on the pan.

Keep the water in the pan boiling so that there may be plenty of steam, and cook from twenty to thirty minutes, until the fish loses its clear, transparent appearance and looks quite white.

If the pieces are thick it is better to turn them while cooking. The liquid that is on the plate when the fish is cooked is the juice from the fish, and should be served with it. Serve the fish with a little plain cold butter, and a piece of plain bread or toast.

Stewed Fish

1 filleted fish, whiting, haddock, sole, plaice.
1 tablespoonful bread crumbs.
½ gill cold water.
1 gill milk.
½ oz. butter.
1 teaspoonful chopped parsley.
White pepper.

Wipe the fish with a damp cloth and cut it into small neat pieces. Rinse out a lined saucepan with water, and place the pieces of fish at the foot.

Sprinkle over them a little salt and white pepper, pour in the milk and water; put the lid on the pan, and let the fish cook slowly by the side of the fire until it is ready, which will be in about fifteen minutes. Do not overcook, or the fish will be hard.

Lift out the pieces of fish on to the plate on which they are to be served and keep them hot.

Add the bread crumbs and the butter to the water and milk in the pan. Stir over the fire for a few minutes until the bread crumbs swell and thicken the sauce.

Sprinkle in the parsley and then pour this sauce over the fish.

Baked Fish

¼ lb. uncooked fish.
2 tablespoonfuls bread crumbs.
½ oz. butter.
1 egg.
½ gill milk.
Pepper, salt, and a little lemon juice.

Grease a small pie dish with a little of the butter. Have the fish free from skin and bone, and cut it into neat pieces.

Lay half of these pieces at the foot of the pie dish, sprinkle over them a little white pepper, salt, and a squeeze of lemon juice, and then put on a layer of bread crumbs. Next, put in the rest of the fish, seasoning, and more crumbs.

Beat up the egg in a small basin, add the milk to it, and strain this into the pie dish. Put the rest of the butter in small pieces on the top, wipe round the edges of the pie dish, and bake in the oven until nicely browned.

Soups.—The following recipes given for soups are those in which the ingredients are not too rich for the digestive capacity of the convalescent. They form a delightful change after the monotony of the beef teas and extracts. These recipes are very useful for the dietary of the aged (see p. 490).

Broth—Mutton, Chicken, or Veal

1 lb. neck or knuckle of mutton or veal, or 1 chicken.
2 pints cold water.
1 dessertspoonful of rice.
1 teaspoonful chopped parsley.
1 teaspoonful salt.

Wipe the meat well with a damp cloth. Cut into small pieces and remove it from the bone, and take away as much fat as possible. Put meat, bones, water, and salt into a clean lined pan, put on the lid, and bring it very slowly to the boil.

Remove with an iron spoon all the scum that rises. Simmer slowly for four hours, skimming when necessary.

When cold remove all the fat. Return it to a saucepan with the rice well washed. Allow it to cook again for twenty minutes until the rice is soft. Add the parsley at the last and it is ready for serving.

Tapioca (Groult's) or arrowroot may be used for thickening instead of rice.

Rice Soup

1 pint of mutton, veal, or chicken broth.
1 tablespoonful of Patna rice.
Yolk of an egg.
1 tablespoonful of cream.

Strain the broth and remove all fat from it; put it into a clean saucepan with the rice well washed, and boil until the rice is perfectly soft. Then rub all through a fine sieve, rinse out the pan, and return the soup to it.

Beat up the yolk of egg and cream with a fork, strain them into the soup and stir carefully over the fire until thoroughly hot, but it must not be allowed to boil.

Tapioca or sago may be used instead of rice to thicken with.

Also three tomatoes might be boiled with the rice, and then rubbed through the sieve. This would make tomato soup.

Rabbit Soup

1 rabbit.
1½ pints cold water.
½ pint milk.
½ oz. butter.
½ oz. flour.
A few pieces of parsley.
1 bay leaf.
6 peppercorns.
½ teaspoonful of salt.

Wash and clean the rabbit well. Let it lie in salt and water for half an hour. Lift out, dry it, and cut into joints. Cut the flesh into small pieces and chop the bones.

Put all these into a saucepan with cold water, with bay leaf, parsley, salt, and peppercorns. Bring to boil and skim well.

Simmer slowly from five to six hours; then strain through a fine sieve. Put the meat into a mortar; pound it well with a little liquid, and rub it through a wire sieve.

Rinse out the pan; melt in it the butter; add the flour, and mix till smooth. Then add the sieved meat, soup, and milk, and stir until boiling. Boil for ten minutes.

Game Soup

1 bird (grouse, pigeon, woodcock, partridge), or the remains of game.
¼ lb. lean juicy meat.
1½ pints cold water.
½ teaspoonful arrowroot.
A pinch of celery seed, pepper, and salt.

Wipe the game and meat with a cloth; shred the meat as for beef tea; cut the game up into neat pieces. Put all these into a lined saucepan, with cold water and seasoning; bring slowly to the boil, and skim thoroughly. Allow the soup to simmer for four hours. Strain and allow to cool, and then remove the fat.

Mix the arrowroot with cold water; add this to the soup. Boil for two or three minutes, until the arrowroot turns quite clear.

Fish Soup

Small haddock or whiting, or piece of cod.
½ oz. butter.
½ oz. flour.
3 gills or 1 pint cold water.
1 gill of milk.
1 yolk of egg and ½ gill of cream.
1 teaspoonful finely chopped parsley.

Wash and scrape the fish very clean; see that there is no black skin lining the inside part. Remove the eyes.

Cut the fish across into several pieces, and put them in a lined saucepan.

Cover the fish with cold water, and add the salt. Bring to the boil and skim.

After the soup has boiled for a few minutes lift out a few nice little pieces of fish free from skin and bone, and reserve them for serving in the soup at the end.

Allow the rest to simmer slowly for ¾ to 1 hour. Then strain through a wire sieve, and rub some of the white pieces through.

Rinse out the pan the soup was cooked in. Melt in it the butter; add the flour, and mix these two smoothly together, being careful they do not brown. Then pour on the soup that has been sieved, and stir until boiling.

Beat the yolk of egg and cream and milk together; and, when the soup is off the boil, beat these ingredients into it; then strain through a fine strainer, stirring all the time. Do not let the soup boil after the yolk is added, or it will curdle. The pieces of fish that were reserved and the chopped parsley are now added.

MEATS

Of this class of food poultry, game, and rabbits are the most easily digested. They can be served in the usual manner of roasting, boiling, and stewing. A few extra methods of preparation are here given.

Tripe and sweetbreads form most excellent dishes for persons of weak digestion, and only require to be more thoroughly known to be in common use (see pp. 485, 486).

Broiled Chicken

1 small chicken.
1 oz. butter.
Pepper and salt.

Prepare a young chicken for roasting, split it down the back, and lay it open, and take only half at a time. Rub the piece of chicken over with a little butter to keep the skin from cracking, and season with pepper and salt.

Grease the gridiron and make it thoroughly hot. Lay the chicken on it with the cut side down to begin with; broil either on the top or before a clear fire for about half an hour. When cooked lift on to a very hot plate, and rub the rest of the butter over it. Serve with rolls of bacon round it.

If broiling an older fowl it must be partially cooked first either by boiling or roasting.

Chicken Soufflé

Breast of chicken.	Flour, ½ oz.
Cream, 1 gill.	Salt, } to taste.
Egg, 1.	Pepper, } to taste.

Skin the breast of the chicken; chop it finely; pound well in mortar; melt the butter; stir into the flour, and a tablespoonful of cream. Let it come to the boil; pour it over the pounded chicken; add seasoning, pound together, and rub through a wire sieve.

Switch the remaining cream until stiff; mix gently with other ingredients; butter some cups; half fill with mixture; cover the cups with kitchen paper, and set in stewpan half full of boiling water. Steam for fifteen minutes. Serve with white sauce.

The boiling water should only come half-way up cups while steaming.

Chicken or Veal Panada

¼ lb. breast of chicken or ¼ lb. fillet of veal.
1 teaspoonful cold water.
1 tablespoonful cream.
A pinch of salt.

Wipe the meat and cut it into small pieces, free it from fat and skin. Put into a cup with a pinch of salt or cold water. Tie over it a greased white paper, and steam slowly for 1 to 1½ hours. Then lift it out, place the contents of the cup into a mortar; pound well and rub through the sieve.

Put the sieved mixture into a pan, add the cream, and heat thoroughly. This may be served on a piece of toast or even taken cold.

Ragout of Fowl or Rabbit

Take the wings and breast of a fowl or the back and legs of a rabbit; soak in hot water for five minutes, then put into a jar with salt and mace or peppercorns.

Cover with milk; cover the jar closely, set in an oven, and bake for three hours. Take out the meat. Pour the milk into a basin to cool, so that the fat may be easily removed. Warm in a cup set in hot water when required, or warm a little of the meat in the milk, and serve with thin toast or water biscuits.

Tripe, Methods of Preparing.—The best varieties of tripe are those known as the "blanket" (because it has a folded appearance), the dark variety known as the "monk's hood,"—these are best for invalids on account of being more tender. The other sorts are known as the "book" and the "honeycomb."

If tripe is properly prepared it is a most delicately flavoured and easily digested article of food, and should be in much more constant use than it is at present. It requires, however, very careful cleaning and boiling. In Scotland we are unable to buy prepared tripe at the butchers, so it is necessary to understand the whole process. Tripe sold in England has generally had a preliminary boiling.

First wash and scrub in several waters and scrape it with a knife, pulling away any pieces of fat from it.

Cut it into pieces and put it into a clean saucepan with cold water to cover it, and bring it to the boil. This is called blanching, and is repeated until the water in which the tripe is boiled loses all its heavy smell. This may require being done four or five times. Then rinse the saucepan well; put in the tripe with cold water to cover it, bring to the boil, and cook *slowly* for ten to twelve hours. If cooked too quickly the tripe will get hard. When sufficiently cooked it ought to be so tender that it will pull easily to pieces when you try it with the fingers. Pour it out into a basin and cover it with the liquor in which it was cooked. This prevents it becoming hard and dry. It can then be made up in any way you wish.

The water in which tripe is boiled should never be thrown away, as it contains a certain amount of nourishment. It is sometimes served as an invalid jelly.

The tripe can now be stewed in various sauces for fifteen minutes.

1. Tripe stewed in white sauce and onions, and served with sippets of toast.

2. Tripe stewed in a thick tomato sauce and served with little rolls of bacon and croquettes of fried bread.

3. Tripe heated in a good curry sauce and served with a border of rice.

4. *Scalloped Tripe.*—Lay a layer of prepared tripe in a buttered fireproof china dish, then a layer of chopped mushrooms and seasoning such as pepper and salt. Add a gill of brown stock, and on the top sprinkle bread crumbs with little pieces of butter. Place in oven to get thoroughly hot and brown.

5. *Baked Tripe.*—Grease out a small pie dish, and put the tripe with thin square pieces of bread and butter into this in alternate layers. The last layer should be bread with the buttered side up.

Beat up an egg in a basin until it is frothy, add it to half a gill of tripe liquor, and season with pepper and salt. Strain this into the pie dish and then wipe round the edge of the dish with a cloth.

Let the mixture stand for ten minutes until the bread gets thoroughly soaked.

Bake in a moderate oven for about fifteen minutes until nicely browned. Serve hot.

Sweetbreads. — This is another article of food that must be nicely prepared, or it is quite disgusting. The difficulty is to clear off all the connective tissue and fat from between the lobules.

Soak the sweetbread in cold water for one to two hours. Then put it into a saucepan with cold water to cover it. Bring to the boil and let it boil for five minutes. Then lift it out into a basin of clean cold water to cool it. This preserves the colour of the sweetbread. Then with great care remove from it all the fat and skin, pulling them off with the fingers. It is now ready for cooking.

Braised Sweetbreads.—Prepare the sweetbreads as above. Braise them for 1½ hours—that is, place the sweetbreads unbroken into a saucepan containing a little onion, carrot, turnip, celery, savoury herbs and seasoning; add a little meat stock, just enough to cover the meat, cover with paper and a well-fitting lid.

At the end of the time take them out, brown in the oven, and serve on a piece of fried toast, or a bed of mashed potatoes. Pour a good sauce round them, such as sauce Italienne.

Sauce Italienne

1 oz. flour.
1 oz. butter.
1 bay leaf.
Onion and spring parsley.
1 gill sherry.
½ pint second stock.
Pepper and salt.

Melt the butter, fry the herbs and onions and flour a good brown colour. Add the sherry and allow it to cook a few minutes, add the stock when boiling and allow to simmer for an hour. Strain the sauce before pouring round the sweetbreads.

Stewed Sweetbreads

1 heart sweetbread.
1 gill of white stock.
1 teaspoonful arrowroot.
1 tablespoonful cream.
White pepper and salt.

Break the prepared sweetbread into small pieces of equal size, put them into a small lined stewpan with the stock, put on the lid, and allow it to simmer very slowly until tender.

When cooked lift the pieces of sweetbread out, place them on a neat square of toast on a hot dish, and keep them warm.

Break the arrowroot in a small basin with a little cold water, and add it to the stock in the pan. Stir over the fire until boiling, and boil for five minutes so as to cook the arrowroot.

Add cream and season to taste. Pour the sauce over the sweetbread, and serve very hot.

Lambs' sweetbreads are very delicate, and can be cooked in this way.

See also recipe in diabetic feeding, p. 500, for sweetbreads and tomatoes.

Minced Beef

½ lb. best rump steak.
½ pint cold water.
Pepper and salt.
Sippets of toast.

Choose the beef and select a nice juicy piece.

Either get the butcher to mince it, or after removing all fat and connective tissue put it through the mincing machine.

Never buy mince from the shop for an invalid, there is always too much fat in it.

Take a small lined stewpan; put the minced beef into it with half the water and the seasoning.

Put the pan on the stove and pound the meat well until it loses its raw appearance.

Then add the rest of the water, simmer gently by the fire for twenty minutes. Do not allow the meat to cook too quickly or it becomes hard. Remove any grease that may rise on it, and serve very hot.

Garnish the dish with some neat sippets of toasted bread.

Meat Juice Mince

½ lb. best rump steak.
1 small piece of butter.
Pepper and salt.

Take the meat and rub through the hair sieve until all the red juicy part has gone through, scrape the bottom of the sieve.

Melt a very small piece of butter in a small frying-pan, toss the meat juice in it for three or four minutes until it loses its red colour. Flavour and serve with toast. This looks just like mince, but as none of the fibre is present it is very digestible. This meat juice mince can be made more easily digestible by omitting the butter, and adopting the following method:—Take the scraped juice and add a tablespoonful of beef tea or simple stock, and stir in an iron pan for three or four minutes, when the juice granulates. If an enamel pan is used the meat has a very unappetising appearance.

Beef Quenelles

¼ lb. beef steak.	Salt and pepper to taste.
Bread crumbs.	1 egg.

2 tablespoonfuls stock.

Pound the beefsteak, half a teacupful of bread crumbs, salt and pepper, egg and stock, well in a mortar, rub through sieve, shape with tablespoons, and poach in shallow pan for ten minutes in boiling water.

Serve with sippets of toast and pour a gravy made of thickened beef tea round the quenelles.

VEGETABLES

As the nutritive value and digestibility of vegetables depend largely on their careful preparation, a short account of the best methods of preparing many of them is here given:—

Spinach.—One of the most easily digested vegetables if properly cooked.

The preliminary difficulty in thoroughly cleaning spinach and the removing of the tough stalks are the two important points in the preparation of spinach. It also reduces enormously in cooking, and 1 lb. will make a very small dish.

Double the leaves lengthways and strip off the stalks. Then wash the spinach thoroughly in several waters until all the grit is removed.

Handle it as lightly as possible, as touching it too much causes the leaves to lose their crispness.

Put it into a saucepan without any water except that which adheres to the leaves; sprinkle it with salt and put the lid on the pan.

Spinach is the only green vegetable which is cooked with the lid on the pan, as no water is used. Were the lid off the spinach might burn from the evaporation. Cook until it is quite tender for stirring with a spoon, twenty to thirty minutes.

When ready drain off the water in a fine wire sieve with a basin below. Then remove the basin and rub the spinach through the sieve on to this; scrape the sieve well beneath. Return the spinach to a saucepan with a small piece of butter, pepper, and salt, stir over the fire until thoroughly hot, add a squeeze of lemon juice or cream.

This can be eaten with small pieces of toast of fried bread.

If served with a poached egg on it, it will make a nice dinner for a delicate person.

Boiled Lettuce.—Wash a couple of lettuces thoroughly and remove any discoloured leaves, and let them lie in cold water for a short time.

Drain the water off, and put them in a pan of boiling water and a little salt.

Boil quickly for about twenty minutes, when they should be quite tender. Keep the lid of the pan off.

Remove any scum that rises.

When ready, drain and chop up the leaves on a board.

Return to the saucepan with a small piece of butter, pepper, and more salt if required.

Tomatoes.—These have most flavour when baked, but can be boiled or stewed.

Wipe them first and remove the stalks.

Put them on a greased baking-tin or fire-proof dish, a little pepper and salt, and cover with a piece of greased kitchen paper. Bake for ten minutes until they feel soft. Lift on to a clean hot dish and serve.

Steamed Asparagus.—Trim the asparagus, and steam by putting it in a jam pot nearly filled with boiling water, placed in a large saucepan half full of boiling water and lightly covered.

The asparagus will take nearly an hour to cook in this manner.

Serve with a sauce made of 1 oz. of melted butter, 1 tablespoonful of cream, the yolk of an egg, and 5 drops of lemon juice. Stir in mixture for a few minutes over the fire until thoroughly warmed.

Celery, Sea-Kale, and young Leeks.—For celery, choose it when fresh and crisp, remove the coarse outside leaves as they can be used for flavouring.

Put away the root, separate the stalks, wash and brush them well in cold water, and scrape off any brown or discoloured parts with a knife, cut the stalks equal lengths, tie them together with tape, string would cut through, and throw them into a basin of clean cold water, and allow them to soak in this for a few moments before cooking.

To cook. Put into a saucepan of a freshly boiling mixture of equal parts of milk and water, and a little salt. Boil with the lid off for about half an hour, when it should be quite tender.

Drain well, remove the tape and serve on a small piece of toast. This is not meant to be eaten, but it is for the purpose of more thoroughly drawing the moisture of the vegetable.

All these vegetables can be served with a little well-made white sauce served round them. This sauce, unless properly made, is much better away, as butter sauce, as commonly served, is a compound of uncooked flour and milk.

White Sauce

¼ oz. butter.	1 gill of milk.
¼ oz. flour.	A squeeze of lemon juice.

A pinch of salt.

Take a small lined saucepan: rinse it out first with cold water to prevent the sauce sticking to the foot of it, and melt in it the butter over the fire, being careful it does not brown. Then add the flour, and mix with a wooden spoon until smooth, cook it for a minute or two over the fire to give it a glossy appearance. Draw the pan to the side of the fire, add the milk or fish stock, and then stir constantly over the fire until boiling.

Boil for two or three minutes in order to thoroughly cook the flour, and season to taste.

Cauliflower.—A young cauliflower with a firm close head. Trim off the thick part of the stalk and nearly all the leaves. Split the stalk in opposite directions that the water may get in and cook it well. Wash in cold water, and let it lie in fresh water and 1 teaspoonful vinegar to draw out any insects.

Have on the fire a deep saucepan three-quarters full of briskly boiling water, add salt to it, and put the cauliflower in head downwards.

Let it boil from twenty minutes to half an hour, until the flower feels tender, but is not broken up. Remove the scum as it rests. This besides being dirty would discolour the head. When ready, lift it out and drain for a minute or two on a sieve, and then serve with the white sauce, or white sauce and grated Parmesan cheese browned as on p. 498.

Onions.—Spanish onions are by far the best for eating as a dish, the flavour being not so strong.

When onions are plain *boiled* they are best served on dry toast without any sauce. A large Spanish onion takes about three hours' boiling to become tender.

Baked.—Place one or two Spanish onions in a baking-tin with a little butter: baste occasionally. When finished they should be of a nice brown colour. They will also require about three hours' baking.

Stewed.—Place a large Spanish onion in a saucer at the bottom of the saucepan, and put sufficient equal parts of milk and water to reach the edge of the saucer. Keep the lid of the saucepan on tight, and let it steam for about 3½ hours until it is quite tender. The water from the onion will prevent the necessity of adding fresh water. Serve it with the milk in which it has been stewed.

Plain Boiled Rice.—Well wash some Patna rice in several waters until the last water looks quite clean. If there is a pot for steaming the rice it is best to use this, but if not, boil the rice in a saucepan of boiling water containing salt, which is in the proportion of one teaspoonful to the quart.

Boil quickly with the lid off, stirring it frequently with a fork to prevent it sticking to the pan. Cook from ten to fifteen minutes until the grains will rub down easily when one is tested between the finger and thumb.

Strain through sieve or strainer, and finish the cooking by drying it, either by putting it into the saucepan by the side of the fire, or putting it on to a plate in a moderate oven.

While drying stir lightly with a fork every now and then to keep the grains separate.

N.B.—The water in which the rice has been boiled contains the best part of the rice, so it should not be thrown away, but kept for the stock-pot.

Boiled Macaroni.—Break the macaroni into short lengths, and throw it into a saucepan of freshly boiling water with salt in it.

Boil quickly with the lid off the pan until it has thoroughly swelled and is tender. Stir occasionally to prevent it sticking. The time depends on the variety of macaroni—the large pipe will take about half an hour, the small much longer.

Keep it well covered with water.

When ready, drain, and this may either be served plain with meat, or it may be put back into the saucepan with enough stock to cover and allowed to stew for half an hour.

The pulp of a fresh tomato rubbed through a sieve may also be added to this.

Food for the Aged

Owing to the failing intestinal activity incident to the later years of life the digestive tract in old people has to be treated with more consideration than obtains in earlier years. This is especially true of those subjects whose "old age" has been induced by a manifest organic weakness in the kidney, stomach, heart, lungs, or other viscera, but it is also applicable to others in whom no such manifest organic disease exists.

The old adage "once a man and twice a child" applies very aptly to the feeding of the aged. It is impossible to lay down specific directions appropriate to the numerous conditions comprised under this heading, and all that will be attempted is to indicate a few guiding rules as to the choice and preparation of the most suitable forms of nourishment.

It may be stated as an invariable rule that the intake of food should be diminished as the general bodily activity decreases, and further, that red meats and rich oily dishes should give place to white meats and simple farinaceous foods. All foods must be in a form admitting of easy mastication.

Milk and Cream.—Cream is a capital substitute for other forms of fatty foods to which the patient has been previously accustomed. Milk should always form a large portion of the dietary if it is digested. The tendency should be to increase the intake of milk as the vegetables are diminished.

If milk taken alone is too constipating add a little saline water, such as Vichy or Vals. It is best always to take the chill off the milk before giving it to old people. Whey (p. 464) can be used as a substitute for milk, and with the addition of cream it forms both a pleasant as well as a nourishing food.

Milk is also combined with the various forms of gruels and farinaceous puddings.

Cocoa and milk and coffee and milk are also very good, and if these prove indigestible the peptonised brands before mentioned (p. 472) may be tried instead.

Soups.—These, when made simply, are all permissible, but rich consommés, hare soup, turtle soup, etc. throw too much strain on the digestion to be advisable.

Chicken, veal, and beef teas (p. 474) may be used as beverages, and are most useful for giving to the patient last thing before retiring to bed, or a cup of "tea" left beside the bed to take if the patient is wakeful. In addition to the soups already recommended for convalescence the following slightly richer recipes are commendable. They are not too rich, and yet contain sufficient nourishment for them to form an important item in the diet.

Brunoise Soup

1 carrot, young.
½ turnip, young.
2 leaves of celery.
Flower of small boiled cauliflower.
1 onion.
1 oz. butter.
1 pint of water.
1 pint milk.
1 teaspoonful of salt and pepper.
2 oz. of stale bread, toasted.

Stew the ingredients, except the toast, together for one hour, then break the toast in pieces, add it to the rest, and stew all together for another hour. Pass all through the sieve and return to the stewpan to get hot.

Lentil Soup

Wash the lentils thoroughly in cold water and add the lentils to water in the proportion of ¾ lb. lentils to a gallon of water or of second stock. Add pepper, salt, onion, turnips, carrot, and celery. Boil all for three or four hours. Pass through fine wire sieve or colander, put on a few minutes to heat, and send to table with toast cut in dice. A little curry powder may be added if desired.

This soup is somewhat flatulent to those of weak digestion, but if made without the additional vegetables it will be found very acceptable to the most delicate stomach.

Pea soup served with dried mint, and haricot bean soup, can be made in the same way.

Potato Soup

1 lb. potatoes.
1 leek.
1 onion.
1 oz. of butter.
1 pint milk.
1 pint water.

Stew the potatoes, put them with leek, onion, and butter into a pint of boiling water in a stewpan. Boil until the vegetables are soft, then pass them through a sieve, adding a pint of hot milk to help the union. Put them all into the stewpan until it boils, then serve.

Serve with dice of fried bread.

Rice and Tomato Soup

1 pint of mutton, veal, or chicken broth.
1 dessertspoonful Patna rice.
3 fresh tomatoes.
A little salt.

Put the broth into a clean lined saucepan, with the rice well washed, and the tomatoes wiped and cut in slices.

Boil slowly for half an hour, stirring occasionally.

Then rub through a hair or fine wire sieve. Heat again, season to taste, and it is ready for serving.

FISH

Fish are all allowable for old people. The oily fishes—such as salmon, herring, mackerel—are the only ones that are apt to disagree with digestion, unless taken in small quantities.

In addition to the usual methods of boiling, frying, and baking fish, already described, a variety of dishes may be made from this class of food that are found to be appetising as well as quite satisfying.

Boiled Cod or Haddock Roes.—Wash the roe well; then weigh it and tie it up in a piece of muslin or a pudding cloth. Put it into a saucepan or fish kettle, with enough boiling water to cover it. Add 1 teaspoonful of vinegar and ½ teaspoonful of salt to each quart of water.

Boil slowly, allowing twenty minutes to the pound, or until the roe feels quite firm to the touch. Lift out, drain well, and serve as much as will be required at one time.

It can be served with plain cold butter or white sauce.

Grilled and Fried Roe.—The roe must be boiled first. When it is cold cut into slices half an inch thick with a sharp knife.

To grill, grease the grill or gridiron with a little butter, and make it quite hot. Lay the slices of roe on it and broil in front of a clear fire for five or seven minutes. Serve this very hot.

To fry, melt 1 oz. butter in a frying-pan when smoking hot; put in the pieces of roe. Fry them a nice brown colour on both sides. When ready, lift out and drain on a piece of double paper. Serve very hot.

Fish Baked in Batter

¼ lb. uncooked fish, haddock, or sole.
1 oz. flour.
½ gill milk.
1 egg.
½ oz. butter.
Pepper, salt, and lemon juice.

First make the batter; rub the flour through a wire sieve, and make it free from lumps, and put it into a basin. Beat up the egg with a fork and add it to the flour; beat with a wooden spoon until quite smooth and free from lumps. Then add the milk and beat for a few minutes longer. The more the batter is beaten the lighter it will be.

Have the fish free from skin and bone, and cut into small pieces. Lay these at the foot of a small greased pie dish, and season with pepper, salt, and a squeeze of lemon juice. Pour the batter over, and put the rest of the butter in small pieces on the top.

Allow this to stand for a few minutes before cooking; this gives time for the flour in the batter to swell, and it will be lighter when baked.

Bake in a quick oven from twelve to fifteen minutes until well risen and nicely browned. Serve at once, as the batter quickly falls.

Fried Haddock and Tomatoes

1 dried haddock.
2 tomatoes.
1 small onion.
1 oz. butter.
Pepper, salt, and parsley.

Soak the fish for three hours; then skin it. Take out all the bones, and break up the fish into flakes. Slice the onion and tomatoes; chop up the parsley; sprinkle with pepper and salt, and cook all in the butter until quite soft; then add the fish, and cook for ten minutes longer.

Dish up in a border of boiled rice or mashed potatoes.

Fish Soufflé

¼ uncooked fish.
½ oz. butter.
½ oz. flour.
½ gill of fish stock or milk.
2 eggs.
Pepper, salt, and lemon juice.

First make a panada with the butter, flour, and fish. This is done by melting the butter in a small saucepan, adding the flour, and mixing until it is smooth with a wooden spoon. Then pour on the milk or fish stock, and stir until the mixture is thick and free from lumps, and leaves the sides of the pan quite clean.

Scrape the fish down finely with a knife. Put the panada into a mortar with the fish, seasoning, and yolks of eggs. Pound well together and rub through a sieve.

Beat up the whites of eggs to a stiff froth and stir lightly into the fish mixture with an iron spoon. Pour into a greased basin which should be only half full. Cover with a greased paper. Steam for twenty minutes. When firm, lift it out and turn out on a hot plate.

Curried Fish.—A very nice way of having fish is to curry it and serve it with a border of rice. The following ingredients should be put into the curry.

1 lb. fish. 1 apple or stick of rhubarb.
2 ounces of fat or butter. 1 small onion.
1 tablespoonful of curry powder. 1 tablespoonful of flour.
Salt and pepper. A teaspoonful of lemon juice or vinegar.

Scolloped Fish

¼ lb. cold cooked fish. Bread crumbs.
1 ounce butter.
Pepper and salt.
Fish stock or milk.

Butter a scollop shell of fireproof china; sprinkle on it a layer of bread crumbs; then a layer of fish broken up into pieces; some pepper, salt, and piece of butter. Cover this with more bread crumbs and bits of butter, and pour on a few drops of fish, liquor, or milk. Bake ten minutes.

Kedgeree

Cold fish, ¼ lb., or dried haddock.
Rice, boiled, ¼ lb.
Butter, 1 ounce.
Egg, 1
Cayenne pepper; salt.

Wash and boil the rice; boil the egg very hard; break the fish into pieces, and carefully remove all the bones; take the egg, break off the shell, and cut the whites into small square pieces.

When the boiled rice is dry, melt the butter in a stewpan, add the rice, then the fish, the chopped white of egg, Cayenne pepper, and salt. Mix them well together and serve on a hot dish, and sprinkle the yolk of egg over it.

Macaroni and Fish

¼ lb. macaroni, well boiled.
¼ lb. cold boiled fish.

Mix the macaroni and the cold boiled fish well together with pepper and salt, half a pint of fish stock or chicken broth, and 1 ounce of butter.

Put the mixture in a flat china dish, sprinkle bread crumbs and a little piece of butter on top; when the mixture is quite hot and brown it is ready to serve.

Vegetables and Fruit.—Old people are very apt to give up vegetables owing to their supposed flatulent properties. This practice is serious, for if persisted in, some minor symptoms of scurvy not infrequently develop. Everything depends on the form in which the vegetables are administered and on the amount and nature of the ingredients of the meal.

A small quantity of potato should be taken every day, and also a certain amount of well-cooked vegetable. Spinach, stewed lettuce, stewed or baked tomatoes, vegetable marrow and cucumber, boiled, stuffed, or stewed, or served with grated cheese, celery, sea-kale, asparagus, leeks, the flower of cauliflower, large Spanish onions, green peas when young and quite tender, and French beans, are all suitable. Uncooked vegetables, as partaken of in salads, are not very satisfactory, and are very apt to cause flatulence. So also are cabbage, greens, Brussels sprouts, turnips, parsnips, and old carrots. For methods of preparation, see p. 488.

Fruit taken in small quantity is also advisable. It is best to take it cooked, either stewed or baked, and eaten with cream.[1] This gets over the difficulty of eating a crisp apple when the teeth are imperfect.

In stewing fruit, if cane sugar is added by the cook the resultant product is very prone to turn acid. It is therefore better to neutralise the acidity with an alkali rather than to attempt to mask the flavour with sugar. Thus to each pound of fruit add as much bicarbonate of soda as will lie on a shilling. The bitterness of the fruit will be gone and the natural flavours of the coulere will become apparent, which is usually quite sweet enough. If this simple plan is carried out many old people can eat and enjoy stewed fruit without feeling that they will have to suffer from acidity afterwards.

If there is a desire for sweetness saccharine can take the place of sugar. The usual rule, however, will be found to be that the desire for sweetness disappears, and that food with a relish has to be provided instead.

Alcohol.—A small amount of (measured) alcohol with food is often beneficial. A nourishing stimulant to be highly recommended is an ounce of dry cherry brandy mixed with a wineglassful of cream.

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[1] Preferably not later than the mid-day meal.

Cookery in Diabetes

As a special knowledge of cookery is required to enable the physician in cases of diabetes to frame a diet suited to the physiological capacities of the individual and at the same time to make the necessarily restricted diet more readily tolerated. The points of practical importance in the selection and manner and preparation of individual food-stuffs is here appended. For an account of the principles which must guide the physician in forming the diet the reader is referred to the articles "Diabetes" and "Glycosuria." The principles followed in framing these menus have been to make the food as appetising and varied as possible, while the important articles of food, starch and sugar, have been excluded and their place taken by the hydrocarbon fat, so that the body may not lack entirely the "fuel" food.

If the excess of fat in the food should give rise to dyspepsia a small quantity of brandy and water after the meal aids digestion.

First, it is advisable to give a list of the articles that may or may not be eaten, as on this the menus are framed.

Food.

May eat

Butcher meat of all kinds except liver.
Tongue, ham, bacon, or other smoked, salted, dried, or cured meats.
Poultry. Game.
Fish of all kinds, fresh, salted, or cured. Shell fish, except the bodies of lobsters and crabs. Mussels.
Animal soups, not thickened with any starchy materials. Beef teas and broths.
Eggs, dressed in any way.
Cheese. Cream cheeses. Butter. Cream.
Greens. Spinach. Turnip-tops. French beans.[1] Brussels sprouts.[1] Cauliflower.[1]
Broccoli.[1] Cabbage.[1] Asparagus.[1] Sea-Kale.[1] Vegetable marrow. Lettuce. Cucumber. Tomatoes. Mushrooms. Mustard and cress. Watercress. Endive. Spring onions. Leeks. Celery. Rhubarb. Pickles.
Oil. Vinegar.
Savoury jelly. Jellies and custards, sweetened with saccharine. Blancmanges made with isinglass or gelatine.
All nuts, except chestnuts. Olives.

May not eat

Sugar in any form.
Wheaten bread and ordinary biscuits of all kinds.
Toast. Rice. Arrowroot. Corn-flour. Oatmeal. Sago. Tapioca. Macaroni. Vermicelli.
Potatoes. Carrots. Parsnips. Beetroot. Peas. Spanish onions.
Pastry and puddings of all kinds, and honey.
All fruit, fresh and preserved, except lemons and unripe fruit.
Liver.
Oysters.
Cockles and mussels.

Beverages.

May take

Tea. Coffee. Cocoa from nibs. Dry sherry. Claret. Dry Sauterne. Burgundy. Chablis. Hock. Brandy. Whisky and other unsweetened spirits. Unsweetened aerated waters. Milk (limited to 1 or 1½ pints daily).

May not take

Milk, except in limited quantities. Sweet ales. Porter. Stout. Cider. All sweet wines. Port. Tokay. Champagne. Liqueurs. Fruit juices and syrups. Cocoa and ordinary chocolate.

In following out these tables we see that cream has practically to take the place of milk, cream being almost free from lactose. Saccharine and specially prepared glycerine are available as ready and safe sweetening agents to take the place of sugar. Saccharine being intensely sweet is only required to be used in very small quantities. Fish and vegetables are cooked and served with a liberal amount of butter. Salmon, herrings, mackerel, sardines in oil, pâté de fois gras, cream cheeses, and rich sauces made with eggs, oil and cream, are specially useful in providing a fair supply of fatty foods.

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[1] These should be boiled in a large quantity of water.

The various preparations of diabetic breads and biscuits in the market are made from almonds and other nuts, bran, gluten, casein, etc. These can be obtained direct from the makers (Blatchley, London, and Callard and Co., London), or the ground nuts, etc. in the form of flour can be purchased and made up in the patient's own house. It should be noted that all reliable diabetic food-stuffs are expensive.

Almond flour made into biscuits is very satisfactory; the oil in their composition renders them a very desirable food in diabetes. Almonds in a solid form.

Unripe fruits and rhubarb, sweetened with saccharine or neutralised with an alkali, have to take the place of ripe fruit.

The deprivation of starch is the greatest trial to all patients, and to many this restriction becomes so irksome that the strictness of the dietary has to be relaxed. However, there are at the present time a great number of substitutes which can be tried to make bread and biscuits, some totally free from starch, others, again, containing small quantities are apt to cause indigestion, but when broken into a powder the process of digestion is not interfered with. Cocoa-nut, hazel-nut, and pine seeds are also available in powder forms, and these are made into biscuits, which are flavoured in various ways.

Bran flour is another product which, if carefully prepared, is entirely free of starch; this can be bought by the pound and made into bran cakes or bread. Biscuits can be procured, either plain or mixed with almonds, cocoa-nut, etc. These latter are certainly very palatable.

Gluten bread in slices, and dinner rolls, are other almost starch-free articles; they are fairly satisfactory, and are quite edible when eaten with butter.

Gluten flour or farina contains, in good specimens only, a small quantity of starch, about 10 per cent. French gluten farina is one of the purest varieties; the ordinary makes contain 25 to 42 per cent of starch; this can be made into loaves, girdle and oven scones.

A special form of brown bread, which is very like ordinary brown bread, but contains no starch, is now prepared by Messrs. Callard.

Casein biscuits are very palatable.

Soy flour and biscuits are not at all palatable, and in addition contain generally 24 per cent of starch (Kinch). Most aleuront biscuits are almost inedible.

Every medical man should test all these articles to see if they are adulterated with starch. The presence of starch can easily be detected in food by applying a few drops of a solution of iodine in water, when the starch will immediately turn a violet-black.

Torrified or much-toasted bread is recommended by some writers, who seem to think that by evaporating the water from a piece of bread some of the starch is also got rid of; the fact is that well-toasted bread contains nearly twice as much starch and dextrine as equal weight of ordinary bread.

Almond Cake

1 ounce almond flour.
2 eggs.
Salt to taste.

Beat the whites of the eggs to a stiff froth, add the almond flour, and beat well together. Spread out into a buttered baking-tin about half an inch thick, and bake in a moderately quick oven from fifteen to twenty minutes.

The mixture should be done quickly and baked as soon as the ingredients are mixed.

These cakes are much improved by adding a small quantity of gluten flour.

Cocoa-nut or Almond Cakes

4 ounces desiccated cocoa-nut or almond flour.
1 pinch of saccharine.
German yeast.
One egg.
2 teaspoonfuls cream.

The almond flour is mixed into a paste with a little water and the German yeast. Allow the mixture to stand in a warm place for twenty minutes. Then add the egg, two teaspoonfuls of cream, and water sufficient to work the mixture. Divide into cakes and bake in a *quick oven.*

Gluten bread in slices, cut into small pieces, soaked in butter and toasted or fried, is very palatable, and will be found a useful article in the preparation of many dishes.

Bran Cakes

4 oz. prepared bran flour.
1 teaspoonful of bicarbonate of soda.
5 eggs.
¼ pint of warm milk.
2 oz. butter.

First mix the bicarbonate of soda with the bran flour, then beat up 2 oz. of butter in a warm basin, shake into it the dry ingredients, beating with a spoon all the time. Next beat up the five eggs in a separate basin before the fire until quite warm, and stir them gradually into the mixture of bran flour, soda, and butter. Beat up all well for ten minutes, and add gradually the warmed milk. Place in buttered tins, and bake in a brisk oven for about ten minutes. The cakes are done when they will turn out of the tins quite easily. This will make about five cakes the size of buns. The cakes can be cut into slices, buttered, and toasted.

Even more palatable cakes can be made with an equal mixture of bran and almond flour. The bran reduces the excessive richness of the almond flour and gives a pleasant flavour.

Bran Bread

½ lb. bran (prepared).
2 oz. almond flour.
3 oz. butter.
6 eggs.
½ pint milk.
2 teaspoonfuls of bicarbonate of soda.
1 teaspoonful of tartaric acid.

Place the butter in a basin and beat it to a cream, then add the almonds and well beat, add the eggs one at a time. Partly mix in the bran before adding the milk. Well mix the whole together, and place it in a well-buttered tin and bake for an hour in moderate oven.

Gluten Bread

1 lb. gluten flour.
¼ lb. of prepared bran.
1 oz. yeast.
1 oz. ground almonds.
2 eggs.
Pinch of salt.

Make a sponge as for ordinary bread and set it to rise. This will take about three-quarters of an hour. Then bake.

The addition of a little ground almonds and two eggs is an improvement.

There are a few forbidden articles of food which, when prepared specially, are quite allowable to this class of patient:—

Sponge cakes, made from almond flour and sweetened with glycerine, are excellent.
Gluten macaroni.
Gluten vermicelli.
Cocoa, when specially prepared.
Marmalade, made with gelatine, glycerine, and orange rind. It is an attractive-looking compound, and a great boon to the diabetic's breakfast table. The flavour is somewhat peculiar, due to sweetness from glycerine.
Chocolate, specially prepared, makes an excellent sweetmeat, and also a very good cup of chocolate. It can be bought unsweetened and flavoured with vanilla or sweetened with saccharine.
Glycerine jujubes can also be procured.

Menus—First Day

Breakfast

Tea and cream.
Whiting fried in butter. Squeeze lemon juice when served. Savoury omelet.
Gluten toast. Brown diabetic bread. Butter.

Lunch

Home-made lemonade sweetened with saccharine.
Vegetable marrow stuffed. Cold ham.
Cream cheese. Almond and bran biscuits. Gluten rolls.

Dinner

Clear soup with mixed vegetables.
Steamed halibut. Slices of lemon.
Fillet of beef and cauliflower.
Green apples stewed, sweetened with saccharine. Cream.
Whipped jelly.
Cheese. Biscuits (made with gluten flour).
Pancakes (made with gluten flour, milk, eggs, sweetened with glycerine).

Stuffed Vegetable-Marrow.—Small marrows make an excellent dish, boiled and stuffed with the stuffing of mushroom forcemeat. The marrow should be first peeled very slightly, then cut long-way into three slices, remove the pips, and the interior filled with the forcemeat. The forcemeat should be made hot before it is placed in the marrow, if not, the marrow will be cooked before the stuffing is heated through. The marrow should be placed in boiling water and boiled until tender; this takes about twenty minutes to half an hour.

Mushroom Forcemeat

1 lb. mushrooms.
Half-teaspoonful lemon juice.
Two hard-boiled eggs.
1 oz. butter.

The mushrooms after being cleaned should be chopped and fried in the butter; lemon juice should be added before they are chopped in order to preserve the colour. Add two hard-boiled eggs to the mixture, and rub the whole through a wire sieve while hot.

When hot this mixture is moist, but on standing gets hard.

Savoury Omelet

Parsley, 1 teaspoonful chopped.
Eggs, 3.
Butter, 2 ounces.

Herbs, savoury
Pepper } for flavouring.
Salt

Melt the butter in a frying-pan, beat up the eggs thoroughly, add a little pepper and salt, parsley and herb, pour the beaten-up eggs into the frying-pan as soon as the butter begins to frizzle, and with a tablespoon keep scraping the bottom of the frying-pan in every part. Go on scraping until two-thirds of the mixture have become lumpy.

Now lift the pan a little off the fire, push the omelet into half the frying-pan. When nearly set hold the pan in a slanting direction in front of the fire, and, as soon as set, slide the omelet from the pan on to a hot dish and serve at once.

This may be varied by addition of a little cooked fish, grated cheese, tomato, ham, crab.

Second Day

Breakfast

Tea or coffee with cream.
Toasted sardines on fried gluten bread. Boiled egg.
Bran bread. Butter. Marmalade.

Lunch

Fried bacon served au purée of spinach.
Cauliflower au gratin with black butter sauce.
Gorgonzola cheese. Gluten dinner rolls.
Slice of almond, pound cake (Messrs. Callard's).

Dinner

Tomato purée.
Steamed sole. Sauce Hollandaise.
Boiled fowl.
Carrageen jelly.
Stuffed tomatoes.

Cauliflower au gratin.—This makes a nice course by itself. The cauliflower should be boiled until quite tender. While boiling remove any scum that rises to the top.

When ready lift it out, drain for a minute or two on a colander, and serve with black butter sauce poured over it. Sprinkle the sauce with Parmesan cheese.

Sauce Hollandaise

Yolks of 4 eggs.
2 oz. fresh butter.
½ gill of cream.
Pinch of pepper, salt, nutmeg.
1 teaspoonful tarragon vinegar.

Place ingredients into small bowl. Stand it into large pan on the fire containing boiling water. Briskly whisk the sauce over the fire until it begins to thicken and presents a rich, smooth, creamy appearance. Great care is required to prevent it from curdling; if, however, this should happen, add yolks of two more eggs.

Stuffed Tomatoes

3 ripe tomatoes.
½ ounce of butter.
1 spring onion.
½ teaspoonful chopped parsley.
1 pinch mixed herbs.
2 mushrooms or a teaspoonful Parmesan cheese.
Crumbs of 1 almond biscuit.
Pepper and salt.

Cut off the stalks, leaving a hole the size of a threepenny bit. Squeeze out the juice and pips. To the juice and pips add the onion chopped, the chopped parsley, mixed herbs, and the mushrooms or cheese. Melt the butter and fry the mixture. Then add sufficient almond biscuit crumbs to make the whole into a moist paste.

Fill the tomatoes with the mixture until they resemble their original shape.

Put a few crumbs and a little piece of butter on the top of each.

Place the filled tomatoes in a baking dish and bake until tender, and moisten with a little oil or butter. Serve in a dish with fried parsley round.

Carrageen Jelly or Irish Moss.—Irish moss is a seaweed, and is collected on the northern shores of Ireland. Its nutritive value is slight; it contains mainly mucilage, and also some iodine and sulphur. It should be well washed and soaked for some hours previous to cooking. The flavour is somewhat peculiar, but delicate, and to some very palatable.

1 oz. Irish moss.
1 quart cold water.
Lemon juice.
Saccharine to taste.

Wash the Irish moss well, and soak it for several hours in cold water. Then put into a saucepan with one quart of cold water and simmer slowly for from four to five hours. Strain, add the lemon juice, and sweeten to taste. Pour into a wetted mould and set aside until cold.

Third Day

Breakfast

Tea and coffee with cream.
Scrambled egg on toasted brown bread. Broiled kippered salmon.
Gluten flour bread. Butter.

Lunch

Cocoa nibs and cream.
Chicken panada and green peas (see p. 485). Potted head.
Imperial cheese. Gluten cracknels. Gluten flour scones.

Dinner

Chicken broth (as p. 483). Thicken with almond flour.
Fish soufflé (p. 492).
Curry and French beans. (The basis of this is cocoa-nut, green apples, or green rhubarb.)
Gluten and almond baked pudding. (Make as semolina pudding, using gluten and almond flour, p. 478.)
Savoury eggs.

Sardines.—There are various forms in the market. The smallest fish are the best. They can be procured smoked or preserved with tomatoes. If rolled in slices of streaky bacon and fried they form a very appetising dish.

Girdle Scones

½ lb. of gluten flour.
½ oz. of butter.
½ teaspoonful of soda.
½ teaspoonful of cream of tartar.
Salt or saccharine to taste.
½ teacupful of butter milk.

Rub butter into the flour and other dry ingredients; make into a soft dough with milk. Form into a scone, or little breakfast rolls, or twists, and bake in oven for fifteen minutes.

Savoury Eggs

3 hard-boiled eggs.
¼ gill of cream.
½ oz. of butter.
1 tablespoonful of cheese, Parmesan.
1 small piece of onion chopped.
Pepper. Salt to taste. Mustard.
1 tomato.

Divide the eggs in half; take out the yolks.

Mix together the yolks, cream, cheese, butter, onion finely chopped; pepper, salt, and mustard to taste. Fill the white parts of eggs with the mixture.

Cut six rounds of brown bread; butter them; on each piece lay a slice of tomato, and on this half of a filled egg. Garnish the dish with cress.

Almond Pudding

2 eggs. ¼ lb. of almond flour. ¼ lb. of butter.
2 tabloids of saccharine dissolved in a tablespoonful of brandy.

Warm the butter, beat in the almond flour and the yolks of the eggs, adding the dissolved saccharine. Whisk the whites to a stiff froth, beat all together. Put into small cake moulds, bake in a quick oven, and serve with a little hot sauce made with dry sherry and saccharine.

Cocoa-nut pudding can be made in the same way.

Fourth Day

Breakfast

Tea, coffee, or cocoa from nibs.
Grilled kidney and bacon. Crab omelet.
Bran bread and cocoa-nut biscuits.

Lunch

Lime juice and aerated water. (Unsweetened Montserrat lime juice.)
Anchovies on fried gluten bread slices; if cold, a spoonful of cream on the top.
Cold meat and tomato salad.
Pâté de foie gras, with bran biscuits.

Dinner

Soup Juliene.
Salmon. Mayonaise sauce and cucumber.
Sweetbreads and tomato.
Junket.
Mushrooms au gratin.

Tomato Salad.—Slice up two perfectly firm and ripe tomatoes. Sometimes it is necessary to remove the core and pips. Add a teaspoonful of oil and two teaspoonfuls of vinegar, and sprinkle on a little pepper and salt.

Mayonaise Sauce

Yolks of 3 eggs.
½ pint best salad oil.
½ gill tarragon vinegar.
Pepper and salt.

In a round basin begin stirring, with a wooden spoon, the yolks of the eggs, pepper and salt, until they are quite mixed. Then add, drop by drop, the salad oil and the vinegar, stirring all the time in the same direction; if added quickly the egg curdles. When finished the sauce must have a creamy appearance.

Sweetbreads and Vegetables.—For the preparation of sweetbreads, see p. 486. The prepared sweetbreads are first stewed in milk, then rolled in slices of fat bacon, and placed in the oven for a quarter of an hour.

The bacon is then removed; the sweetbreads are cut in slices, and grated Parmesan cheese is shaken over them. They are again placed in the oven and braised in a rich brown gravy.

Served on croutons of gluten bread, in the centre of which can be placed stewed tomatoes, green peas, fresh French beans, stewed mushrooms.

Mushrooms au gratin

10 mushrooms.
Piece of onion.
1 teaspoonful chopped parsley.
1 saltspoonful of thyme, fresh.
" " dried.

Pepper, Salt, Lemon juice } to taste.
Almond biscuit crumbs.
1 oz. butter.

Select 10 cup mushrooms about the same size. Peel the mushrooms very carefully without breaking them; cut out the stalks close down with a spoon, and scoop out the inside. Peel the stalks, and chop them up with the scooped-out portion; add the onion, parsley, and thyme. Fry all this in the pan with a little butter. Add sufficient biscuit crumbs to make the whole into a moist paste, and season to taste. Bake in the oven until the mushrooms are quite tender. Serve with some nicely fried parsley round it.

Fifth Day

Breakfast

Tea, chocolate, coffee, and cream.
Findon haddocks, stewed in milk or broiled.
Poached eggs on spinach.
Almond loaves. Gluten cracknels. Butter.

Lunch

Cocoa nibs.
Potted herrings. Stewed cucumber and cheese sauce.
Stilton cheese. Pine biscuits. Toasted gluten flour bread.

Dinner

Purée of lettuce or spinach.
Turbot and tartar sauce. (See *infra.*)
Roast chicken and water-cress.
Blanc-mange.
(See p. 479, using saccharine instead of sugar.)
Tarragon creams.

Stewed Cucumber and Cheese Sauce.—Cut a fresh cucumber in pieces about 4 inches long. Boil until tender.

Pour over the cucumber when cooked the following cheese sauce.

Make the sauce in the same way as sauce Hollandaise (p. 498), omitting the tarragon vinegar, and add 3 tablespoonfuls of grated Parmesan cheese before serving.

Tartar Sauce

2 tablespoonfuls of Mayonaise sauce.
1 teaspoonful chopped parsley.
A small piece of very finely chopped shallot.
1 teaspoonful of mustard.

Mix these ingredients well together and gently heat.

Tarragon Creams

2 eggs.
¼ pint cream.
Pepper and salt.
2 teaspoonfuls chopped tarragon.

Put into a basin 1 white and yolks of 2 eggs, the cream, and a little white pepper and salt. Beat up with a fork until smooth, add a little chopped tarragon.

Butter some little moulds, sprinkle the top with chopped tarragon and truffles.

Pour in the mixture and stand the moulds in a stewpan of boiling water reaching to three-quarters height of the moulds.

When the water boils draw the pan to the side of the stove and poach for about twenty minutes till the creams are set.

Turn out on a warm dish and serve with sauce Hollandaise round them.

Sixth Day

Breakfast

Tea, coffee, with cream.
Cutlets of egg, ham, or crab. Camp pie.
Gluten and bran bread. Butter. Marmalade.

Lunch

Fish roe soufflés.
Smoked tongue and salad.
Cheese, with almond biscuits.

Dinner

Cock-a-leekie.
Whitebait. Lemon sliced. Brown bread and butter.
Roast lamb. Mint sauce. Asparagus.
Gooseberry fool.
Hot caviare on toast. Gluten bread.

Fish Roe Soufflés.—Take six roes of fresh herrings, blanch, and pound them; then flavour with salt, pepper, pounded mace, and nutmeg. Add half oz. butter and yolks of two eggs beaten well together. Whisk the whites of six eggs into a stiff froth. Mix the same with the roes, and bake in ramequin cups for about five minutes.

Serve immediately the soufflés are removed from the oven.

Meat and Hot Water, or "Salisbury" Diet

Great care and consideration are called for, both as to the recommendation of this diet and the best means of giving effect to it. The writer recently saw a patient who came complaining of intense weakness and loss of flesh consequent on a course of treatment lasting nine weeks carried on "by correspondence." The patient had been instructed to take a moderate amount of exercise, including golf, and was greatly surprised to find that not only was exercise impossible, but at times she had hardly strength to stand. Very special precautions are always necessary in stout subjects.

The value of the treatment is undoubted in some cases of indigestion and chronic auto-intoxication associated with it. Further reference is made to it in other articles (*vide* "Gout," "Indigestion," "Obesity," "Sprue").

Meats and Beef Juices.—At first the patient begins with *lean meat of beef* in mince or cakes. However, if the patient is unable to assimilate solid food, Carnick's liquid peptonoids, peptonised home-made beef teas and essences, Valentine's, Wyeth's beef juice, Bovril, Vimbos, Brand's beef juice or meat jelly, may be used instead. Gradually the patient begins to take lean mutton cakes and the white meat of chicken. The white of an egg raw, lightly boiled, or poached, is also allowed.

The following directions will be useful in the preparation of the muscle cakes:—

Lean Meat Cakes.—The beef should be used from well-grown animals, and steaks cut from the centre of the round are the best for this purpose. The beef pulps can be prepared in the following manner:—All the fat, fascia, and connective tissue and bone are removed, and the meat is placed on a board or in a chopping tray, and is shredded down with a blunt knife; the pulp is then scraped together with a spoon, the result is that all the tough fibrinous parts remain behind. The scraped pulp is lightly moulded by the hand into cakes from half to one inch thick, and slowly broiled over a clear fire free from smoke. When cooked, serve on a hot plate with a little butter, and season to taste with pepper and salt. The flavouring may be varied by using Worcestershire or Halford sauce, mustard and horse radish, or lemon juice. A small quantity of celery is also permissible.

Drinks.—The patient is usually advised to drink from one to two pints of hot water (110° to 120° F.) at one and a half hours before each meal, and half an hour before retiring. If the water is drunk slowly the stomach will not be distended, and the patient will not experience any sense of discomfort. If plain hot water is nauseating, it can be made more palatable by the addition of sal volatile, ginger, or lemon juice. When there is intense thirst, a little chloride of calcium or nitrate of potash will make the water a much better thirst-quencher. If constipation is present a teaspoonful of magnesium sulphate can be added to the water.

When the strictness of the diet is first relaxed, the following rice scones, preferably taken hot, are very palatable and easy of digestion.

Rice Scones.—Boil a teacupful of whole rice for 2½ hours. To the rice add quarter of a teacupful of sifted flour, a teaspoonful of baking powder, and sufficient water to make a thick batter; then stir in a pinch of salt and the white of one egg well beaten up. Pour the mixture into well-greased cake-tins and bake in an oven for thirty minutes. To be eaten with butter and more salt if desired.

Oysters are a welcome addition to the monotony of the diet, and may be prepared as follows.

Oysters.—Panned.—Take six deep-sea oysters, place them in a colander and pour cold water over them. Drain for ten minutes. Place the oysters in a very hot iron pan, add salt, pepper, a small piece of butter, and a teaspoonful of meat stock. Cook for a few minutes, and serve the oysters garnished with a thin slice of lemon.

Broiled.—Take six large oysters. Lay them on a board and dry them, season with salt and cayenne pepper. Have a gridiron thoroughly heated, place the oysters on the gridiron and brown them on both sides. Place the oysters on a very warm plate and pour round them a little heated beef juice and a little melted butter.

Iris and Ciliary Body.

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See also ACCOMMODATION, PUPIL

ANATOMY.—The iris is the anterior part of the tunica vasculosa or uveal tract. It is the beautifully coloured and contractile membrane seen through the transparent cornea. By its circumference it is attached to the ligamentum pectinatum and ciliary body. Its anterior surface is free, whilst the posterior surface rests by its pupillary margin against the capsule of the lens; an absence of this support causes the iris to be tremulous—*iridodonesis.* It separates the anterior chamber, between it and the cornea, from the posterior chamber, between it and the lens. Slightly to the nasal side of its centre is the aperture of the pupil, whose diameter, when the iris is resting, varies from three to six millimetres. In thickness the iris is about 0·4 millimetre.

Developmentally, the iris consists of two parts, the anterior portion being mesodermic, the posterior portion ectodermic, in origin. The mesodermic iris is composed of the following layers:—

1. *The anterior endothelium,* continuous with, and similar to, the lining cells of Descemet's membrane and the ligamentum pectinatum. It is broken here and there, where crypts in the succeeding layer exist.

2. *The substantia propria,* consisting of a stroma of connective tissue, containing branching connective-tissue cells, many of which in dark eyes are pigmented. The anterior part of this layer is somewhat condensed and free from blood-vessels; this has been termed the *anterior boundary layer.* Within the stroma are found muscular fibres, blood-vessels, lymphatic tissue, and nerves. The muscular fibres are unstriped, and are arranged to form two definite tracts. One of these, the *sphincter* or *constrictor pupillæ,* consists of a flattened ring around the edge of the pupil, nearer to the posterior than to the anterior surface of the iris. The second muscular tract, the *dilator pupillæ,* is radially placed in the posterior part of the stroma, immediately anterior to the membrane of Bruch. It is composed of delicate spindle cells, which cannot be seen unless the iris is first bleached.

3. *The posterior limiting membrane,* or *membrane of Bruch,* a thin hyaline membrane, continuous with the lamina vitrea of the choroid and ciliary body.

The ectodermic iris is composed of two layers of pigmented cells; (*a*) an anterior layer of spindle cells, the continuation of the pigmented layer of the retina; and (*b*) a posterior layer of polygonal cells, the representative of the remainder of the retina. These layers can only be seen in albino irides, or where the iris has been bleached. Together they form *the uvea.*

The colour of the iris in dark eyes is due to pigment granules in the connective-tissue corpuscles of the substantia propria, especially those in the anterior portion. If pigment is absent from the stroma, and only present in the uvea, the iris appears blue, if comparatively thin, as is the case in children and young adults; where the iris becomes thicker the colour changes to gray. The eyes of newly-born children, even among the dark races, are always blue, since at this age the pigmentation is confined to the uvea. If pigment is absent from both stroma and uvea, the eye appears pink, owing to the choroidal reflex shining through the iris; this is seen in albinos.

The ciliary body is that part of the tunica vasculosa which extends backwards from the base of the iris to the ora serrata. Anatomically, it consists of three parts: (1) an anterior thick portion, which supports the ciliary processes, the *pars plicata* or *corona ciliaris;* (2) a posterior portion, devoid of processes, which gradually thins off into the choroid, the *pars non-plicata* or *orbicularis ciliaris;* and (3) the *ciliary muscle.*

The *ciliary processes* are very vascular folds of a connective-tissue stroma similar to and continuous with that of the iris, and limited externally by the ciliary muscle. They are about seventy in number, and between two and three millimetres long. Lining them, as well as the pars non-plicata, are two layers of cells separated from the underlying connective tissue by the *lamina vitrea.* The layer next this lamina is pigmented, and is continuous with the pigmentary epithelium of the retina behind and the corresponding layer of the iris in front. The inner layer of cells, devoid of pigment, is the sole representative of the remaining layers of the retina, and is called the *pars ciliaris retinæ;* in the iris it is continued as a pigmented layer. It consists of two kinds of cells, one set being cuboidal, granular, and with prominent nuclei; the other set being extremely fine and elongated, so as to form slender fibrils, which unite together and aid in the formation of the suspensory ligament. Secretory tubular glands have been described by Treacher Collins. They exist, for the most part, at the junction of the plicated and non-plicated portions of the ciliary body.

The *ciliary muscle* arises from the fibres of the ligamentum pectinatum opposite the sclero-corneal junction; the greater part of its fibres (meridional) pass directly backwards to be inserted into the external layers of the choroid. The different muscular bundles are separated by small tracts of connective tissue. Other fasciculi (oblique), more loosely arranged, pass inwards to the ciliary processes; these run divergingly, and frequently anastomose with each other. Others, the most internal, on reaching the base of the iris, pass into a direction almost circular, forming the *circular muscle of Müller.* This annular muscle is better developed in hypermetropes than in emmetropes. In myopes, on the contrary, the circular fasciculi are small, the meridional fibres constituting nearly all the muscle.

The fibres are of the unstriped variety; the muscle possesses a network of capillaries and a plexus of non-medullated nerve-fibres, with numerous ganglion cells.

The *blood-supply* of the iris and ciliary body comes from the long posterior and the anterior ciliary arteries.

The two *long posterior ciliary arteries,* arising from the ophthalmic artery, pierce the sclerotic close to the optic nerve, one on either side, and pass forwards between the choroid and sclerotic, supplying neither, till they reach the ciliary region. Close to the posterior border of the ciliary muscle each divides into two branches, which take a circular direction parallel to the equator of the eyeball, and unite with branches from the anterior ciliaries to form the circulus arteriosus iridis major, which lies between the meridional and circular portions of the ciliary muscle.

The *anterior ciliary arteries,* about five in number, come from the muscular and lachrymal branches of the ophthalmic artery; they pierce the sclerotic near the margin of the cornea, and then divide into branches to the ciliary muscle and to the circulus arteriosus iridis major.

The *circulus arteriosus iridis major* gives off branches to the ciliary processes. It also sends branches to the iris, which pass radially towards the pupillary margin. Close to the latter they form an anastomotic ring, the *circulus arteriosus iridis minor,* from which, in the fœtal eye, fine branches pass to the pupillary membrane.

The iridic arteries have thick middle and outer coats, and but little muscular tissue.

The veins of the iris and ciliary body do not exactly correspond to the arteries. Thus the anterior ciliary veins are quite small, and only receive blood from part of the ciliary muscle. They anastomose with the conjunctival veins and with Schlemm's canal. The blood from the veins of the iris and the rest of the ciliary body is returned to the choroidal veins, and so to the venæ vorticosæ.

The *nerves* of the iris and ciliary body are derived from two sources. The *long ciliary nerves* are branches of the nasal branch of the ophthalmic division of the fifth nerve, and are therefore sensory. They supply the ciliary muscle. The *short ciliary nerves,* about fifteen in number, are branches of the ciliary ganglion, being derived from the oculo-motor nerve, and pierce the sclerotic near the optic nerve entrance, passing forwards between this tunic and the choroid. On reaching the ciliary body they form the ciliary plexus, from which fibres pass to the ciliary muscle. Passing on, a second plexus is formed in the iris, from which vaso-motorial branches supply the vessels; muscular branches from the third nerve supply the constrictor pupillæ; sympathetic fibres pass to the dilator pupillæ; while sensory fibres supply the anterior surface of the iris. Ganglion cells are found in the ciliary body, but in the iris their presence is doubtful.

The *lymphatics* of the Iris and Ciliary Body.—There are no distinct lymphatic vessels in the iris, but the sheaths of the blood-vessels contain lymphatic sinuses, and the whole stroma is riddled with lymph spaces. Whether these spaces are in direct communication with the anterior chamber through the crypts described above is not definitely known; they, however, open into the spaces between the fibres of the ligamentum pectinatum, and so reach the canal of Schlemm.

Between the ciliary muscle and the sclerotic coat is a lymph space continuous with that between the lamina suprachoroidea and the lamina fusca, and limited anteriorly by the attachment of the ciliary muscle. It is a common seat of inflammatory exudation or hæmorrhage in cyclitic trouble.

PHYSIOLOGY OF THE IRIS AND CILIARY BODY.—Under this heading the following important subjects are included—the action of light and certain poisons on the pupil; associated movements; the mechanism of accommodation; and the function of the ciliary processes.

Movements of the Pupil.—The iris, with its central aperture, the pupil, serves as a diaphragm to shut off marginal rays; spherical aberration is thus prevented. It also regulates the amount of light entering the eye.

Contraction of the pupil to light is brought about by the action of the sphincter pupillæ, which is governed by a reflex mechanism, of which the optic nerve is the afferent path, and the oculo-motor nerve the efferent path, the centre being situated in the floor of the aqueduct of Sylvius. The method of examining the pupillary light reflexes is as follows: Let the patient be placed in a good bright light, daylight by preference, then cover both eyes with the hands; upon exposing one eye—for example, the right—to the light, its pupil contracts (*direct light reflex*), and the left pupil also contracts (*indirect* or *consensual light reflex*), though to a less extent, being indirectly stimulated through the light entering the right eye; upon exposing the left eye, a further slight contraction occurs in the right eye (consensual reflex) in conjunction with the direct reflex in the left. Consensual light reflex cannot be obtained in those animals whose optic nerves decussate completely. If, after shading the eyes for a few minutes, they are suddenly exposed to a bright light, a slight and temporary rhythmical contraction and dilatation of the pupils ensue—*hippus;* this is very evident in neurotic persons.

Other causes of contraction of the pupil are: (1) stimulation of the optic nerve by agents other than light, as electricity; (2) accommodation of the eye for near objects; (3) the early stages of poisoning by chloroform, alcohol, etc., and nearly all stages of poisoning by morphia, eserine, and some other drugs; (4) deep sleep; and (5) the local application of eserine and other myotics.

Dilatation of the pupil is brought about by the dilator pupillæ, which is governed by a reflex mechanism, just as is the constrictor pupillæ. The dilator pupillæ, as to the existence of which there has been much controversy, has been demonstrated both physiologically and anatomically. It is supplied by sympathetic fibres arising from the cilio-spinal centre in the upper dorsal region of the cord. This centre is subordinate to a higher reflex nerve-centre in the medulla. The fibres leave the cord by the lower cervical and upper dorsal anterior nerve-roots, and, passing into the last cervical and first thoracic ganglia, enter the sympathetic cord, pass upwards to the Gasserian ganglion, and through the nasal nerve enter by the long root the lenticular ganglion, from which they proceed by the ciliary nerves to the iris.

Dilatation of the pupil occurs: (1) when the stimulus of light is withdrawn from the retina; (2) when the eye is adjusted for distant vision; (3) during dyspnœa, powerful irritation of the sensory nerves, violent muscular efforts, in the later stages of chloroform poisoning, and in all stages of poisoning by atropine and certain other drugs; and (4) after the local action of atropine and other mydriatics.

Besides the reflex mechanisms regulating the size of the pupil, a local mechanism is supposed to exist either in the iris itself or in the choroid, where ganglion cells are present. When eserine is applied to the eye, contraction of the pupil is caused whether the third nerve has been divided or not, and with a strong dose it cannot be overcome by stimulation of the sympathetic.

Associated Movements.—Two movements are said to be associated when the special central nervous mechanism used in carrying out the one act is so connected with that employed in carrying out the other that when we set the one mechanism in action we unintentionally set the other in action also. Thus the movements of convergence, accommodation, and contraction of the pupil are produced together during near vision by simultaneous stimulation of all the fibres of the third nerve, and if one of these is in excess, as in convergent concomitant strabismus with hypermetropia, it may be merely necessary to correct the excessive accommodation in order to cure the squint. The nucleus of the third nerve consists of definite component parts; the most anterior portion presides over accommodation; below this is the centre for the action of the pupil; farther down come the cells governing the levator palpebræ and the internal rectus; while most posteriorly the nucleus supplies fibres to the rectus superior, obliquus inferior, and rectus inferior.

The Mechanism for Accommodation. — All are agreed that during accommodation the anterior surface of the lens becomes more convex, but how exactly this is brought about has not been definitely proved. The capsule of the lens is attached by the suspensory ligament to the ciliary processes, and any alteration in its tautness will produce an alteration in the form of the elastic lens. Two chief theories have been brought forward. (1) According to the *theory of Helmholtz*, during the act of accommodation for near objects the suspensory ligament is relaxed. This is chiefly brought about by the annular part of the ciliary muscle, which on contracting causes the ciliary processes to approximate to the axis of the eyeball. Help is also given by the meridional fibres, which, acting on the choroid, draw it and the ciliary body forwards. As a result, the ligament is relaxed, and the elasticity of the lens causes the whole of its anterior surface to become more spherical.

(2) According to the more recent *theory of Tscherning*, the suspensory ligament is tightened during accommodation for near objects. The annular part of the ciliary muscle uses the choroid, to which it is attached, as its fixed point, and draws the ciliary processes towards this, thus tending to cause a posterior displacement of the lens. This is prevented by the rest of the ciliary muscle, which, with the sclero-corneal junction as its origin, pulls on the choroid, causing a forward movement of the vitreous. As a result of these actions, the anterior surface of the lens becomes hyperboloid in form; in the neighbourhood of the anterior pole it becomes more convex, with flattening of the more peripheral zones.

The evidence in support of these two views is considered in the article on "Accommodation" in vol. i.

The Function of the Ciliary Processes.—The ciliary processes have a double function; they support the lens by means of the suspensory ligament, which is partly attached to them, and they take part in the production of the aqueous humour. Exactly what portion of their structure is engaged in this secretion is still a matter of some doubt. The ciliary glands discovered by Collins are considered by him to be the source of the fluid of the eye, but it is doubtful whether they are sufficiently numerous, and it seems better to consider that the processes as a whole are the structures concerned. It has been suggested that the ciliary glands, of whose existence there seems to be no doubt, secrete pigment. They are found in greater number in dark eyes than in blue eyes, and appear to be absent in albinos.

Congenital Abnormalities of the Iris.—Abnormalities in Pigmentation.—Pigment may be completely or almost completely absent from the iris in the condition known as *albinism.* The eyes look pink, on account of the choroidal reflex shining through the non-pigmented iris. For further description of albinism see "Skin, Pigmentary Disorders of."

Pigment may be arranged in the iris not only diffusely, but in small aggregations, giving rise to a mottled appearance.

The iris of one eye may remain blue while that of the other eye is pigmented, becoming brown; a sector of one iris may, again, be of a different colour from that of the rest. These conditions are included under the term *heterochromia.* When the heterochromia is complete, the colour of one may correspond to the colour of the father's irides, and that of the other to the colour of the mother's irides.

Occasionally small masses of pigment are seen at the pupillary margin; these are extensions of the uveal pigment of the iris, and are found congenitally as well as in chronic glaucoma. It is normally found in the horse, and is termed *ectropion of the uveal pigment.*

Abnormalities in Form.—*Irideremia* or *Aniridia* is a more or less complete absence of the iris, a narrow rim alone remaining. It is frequently found in members of the same family, and is often accompanied by choroidal coloboma and lenticular opacities.

Coloboma iridis consists in a congenital cleft in the iris. It is usually directed downwards, or downwards and slightly inwards, and resembles the gap made by iridectomy, except that the constrictor muscle can be seen to line its walls. It varies in extent and frequently occurs in both eyes. It is often accompanied by coloboma of the choroid. It is frequently hereditary, and has been traced through four generations. It has been produced artificially in the offspring of animals, a small sector of whose irides has been caused to become atrophic. Most cases of downward coloboma are probably due to a late closure of the choroidal fissure. On account of this, there is a cleft in the ciliary body, and consequently here either no iris is developed or it is rudimentary. This, however, does not explain the condition of upward coloboma, nor of a double coloboma in the same eye, an abnormality which has been described. These cases are probably due to some irregularity in the production of the secondary optic vesicle, hindering the development of a sector of the iris.

Polycoria is an exceedingly rare condition, in which the natural pupil is divided into two or more apertures by bands of iridic tissue. As many as eight extra pupils have been described, each surrounded by muscular tissue, as shown by the behaviour to atropine.

Corectopia is a congenital misplacement of the pupil. It may be situated quite at the periphery, close to the sclero-corneal junction. It is a rare deformity, and is frequently accompanied by dislocation of the lens.

Persistent remains of the pupillary membrane are sometimes found. The membrane is usually represented by one or more fine filaments which arise from the circulus arteriosus iridis minor (thus being distinguished from posterior synechiæ), and are attached either to the iris opposite or to the anterior capsule of the lens. Occasionally a definite membrane is seen in front of the lens.

Iridodonesis, or trembling iris, is found in cases of congenitally dislocated lens.

INFLAMMATORY CONDITIONS OF THE IRIS AND CILIARY BODY.—Hyperæmia of the iris attends many local inflammatory changes, such as acute conjunctivitis; keratitis, especially if due to a foreign body deeply imbedded in the cornea; other forms of injury; and after intraocular operations, as iridectomy, cataract extraction, etc. Being the first stage of inflammation, its early recognition, combined with prompt treatment, will often cut short an impending attack of iritis. It is characterised by contraction and sluggish action of the pupil; slight discoloration of the iris with indistinctness of its reticulum; engorgement of its vessels, which can often be seen with a strong magnifying lens; media which are not hazy; and a pupil whose outline is clear and which dilates fully and regularly under a mydriatic. The circumcorneal zone is injected, and lachrymation with photophobia is usually present. The treatment consists mainly in local depletion by leeches and the use of atropine.

Inflammatory conditions of the iris and ciliary body are frequently found together; it will be well to consider the symptoms of each separately before seeing what their etiological factors are.

Symptoms of Iritis.—The earliest symptoms are those of hyperæmia of the iris:—1. *The mobility of the iris is diminished;* its movements are either sluggish or lost, on account of spasmodic action of the sphincter pupillæ.

2. *The colour of the iris is altered.* This change is sometimes very slight, but by careful examination with oblique focal illumination there will nearly always be found an alteration in the colour of the tissues surrounding the edge of the pupil. The two irides should be very carefully compared. In many cases the change in colour is very marked, the blue or gray iris becoming yellowish green, whilst the dark brown iris assumes a brownish red or rust colour.

3. *The blood-vessels immediately surrounding the cornea are injected*, forming a pink or deep red ring; there is conjunctival injection, with photophobia and lachrymation.

In addition to these, we have symptoms resulting from inflammatory exudation. *Turbidity* of the iris, due to exudation into its substance, and of the aqueous fluid, ensues. This produces a muddy appearance of the pupil. Pus may collect in the anterior chamber (*hypopyon*), or an iridic vessel may rupture, causing hæmorrhage (*hyphæma*). If the lymph which occupies the pupil becomes organised, we get an opaque persistent membrane, which may be perforated by one or more small apertures. This condition is termed *occlusion of the pupil*. Again, lymph in the posterior chamber may become organised, forming adhesions between the iris and the capsule of the lens (*posterior synechiæ*). If the whole of the pupillary margin is adherent to the lens capsule, the condition is termed *annular posterior synechia;* the anterior chamber is cut off from the posterior chamber (*seclusion*, or *exclusion*, of the pupil). Lastly, the whole of the posterior surface of the iris may be adherent to the capsule of the lens, a condition known as *total posterior synechia*. This, however, is rarely found in absence of cyclitis.

Other symptoms of iritis are *impaired vision* and *pain.* The amount of diminution of vision varies very considerably. With occlusion of the pupil there is very much less sight than with exclusion. Pain may be entirely absent, or may exist in various degrees of severity within the eye and in the surrounding parts. The amount depends largely on the etiological factor.

Symptoms of Cyclitis.—This affection, which, when it occurs without iritis, is usually chronic, with few inflammatory symptoms, is recognised by a somewhat dilated sluggish pupil, a normal-looking iris, slightly raised tension, and by the presence of so-called *keratitis punctata* (or *precipitates*), being a dotted appearance on the lower part of the posterior surface of the cornea. Its pathology will be considered later. Cyclitis, when it accompanies iritis, is much more acute, and is recognised by the following additional symptoms:—(1) tenderness over the ciliary region; (2) increased intraocular tension; (3) vitreous opacities; (4) total posterior synechia; and (5) cellulitis of the eyelids and cheek. It is rare for cyclitis to occur without iritis, the irido-cyclitis usually found being that disease formerly named serous iritis, in contradistinction to plastic iritis with its marked exudation, as described above.

In considering the etiology, pathology, diagnosis, prognosis, and treatment, iritis and cyclitis will be taken together.

Etiology of Iritis and Cyclitis.—In the large majority of cases of iritis and cyclitis the cause is some constitutional taint. It is probably only in the case of a perforating injury that iritis is a purely local affection. Cases of iritis occur, the cause of which cannot be discovered; these, till they are better understood, are termed idiopathic. Hence we have the following etiological classification of iritis and cyclitis:—(1) Symptomatic; (2) traumatic; (3) idiopathic. In addition to these we have (4) secondary iritis, *i.e.* iritis following inflammation of neighbouring structures.

(1) *Symptomatic iritis* occurs as a result of syphilis, rheumatism, gonorrhœa, tuberculosis, gout, and diabetes; it may follow one of the exanthemata.

(*a*) *Syphilitic iritis* is much the most common form of iritis. It appears as a rule within the first nine months of contagion, usually in the fourth or fifth month. It occurs in about four per cent of all cases of syphilis. There are four chief forms, plastic iritis, iritis gummosa or papulosa, gumma of the iris and ciliary body, and irido-cyclitis.

The *plastic* form differs from the other forms in the large amount of exudation, and in the rapidity with which this becomes organised, in showing no tendency to recur when once cured, and in the absence of very severe pain.

Iritis gummosa or *papulosa* appears as a secondary manifestation, and must not be confounded with the next form. It occurs in eighteen per cent of all cases of plastic iritis. Its characteristic feature is the formation of multiple orange or rust-coloured nodules of lymph, either at the ciliary or the pupillary margin of the iris. The ordinary signs of iritis are present. As a result of this form, a broad posterior synechia is very common.

True *gummata* of the iris and ciliary body may occur as a tertiary symptom of syphilis, but are very rare. They usually start at the iridic angle, and cause marked staphyloma. Vision is usually greatly affected, though a case has been recorded (*Ophth. Review*, vol. xvii. p. 238) in which, after correction of the resulting astigmatism, useful vision was restored.

Irido-cyclitis, the fourth variety of syphilitic iritis, is a late secondary or tertiary manifestation, since it may occur several years after the primary infection. It is frequently accompanied by choroiditis.

Hereditary syphilis is occasionally characterised by *congenital syphilitic iritis,* occurring at, or a few hours after, birth. A little later, usually between the ages of six weeks and sixteen months, iritis may develop—*infantile iritis.* Still later, interstitial keratitis may be complicated by iritis, extensive adhesions sometimes resulting. Occasionally cyclitis, with no keratitis, results from hereditary syphilis.

(*b*) *Rheumatic iritis* may follow acute rheumatism, but is more common in the chronic form. It shows a great tendency to recur, the relapses often alternating with the attacks of rheumatism, though in some cases the two are synchronous. It is attended with greater pain and conjunctival injection than the syphilitic form. The plastic exudation, however, is less; there is consequently less change in the colour of the iris, and the adhesions are less extensive and form less rapidly. Sight may be little affected, even after repeated attacks. Both the iris and ciliary body usually are implicated.

(*c*) *Gonorrhœal iritis* resembles rheumatic iritis very closely. It is found especially with gonorrhœal rheumatism, although it may occur in cases where the joints are not affected. Like rheumatic iritis, this form shows a special tendency to relapses, especially after a renewal of the urethral discharge or a return of articular swelling.

(*d*) *Tuberculous irido-cyclitis* is a form of iritis occasionally seen, though, from the rarity of its occurrence, its earlier stages are very liable to be overlooked. It is essentially chronic, and differs chiefly in this respect from all other varieties. This disease is probably always secondary to a primary tuberculous focus elsewhere in the body. In the disseminated variety the pupil is usually contracted and immobile. At first nodulated on its surface, the iris eventually fills up the anterior chamber. Its colour is a dull brick-red mottled with gray, and it appears to be very vascular. The pain and circumcorneal injection are not marked at first, though in the later stages, owing to the supervention of secondary glaucoma, the pain becomes intense and persistent in spite of all palliative measures; chemosis and intense circumcorneal injection accompany this increase of tension. It usually occurs in young subjects. The diagnosis is determined by the chronicity of the affection, by the characteristic appearance of the iris, and by the resistance to all ordinary treatment for iritis. Upon making an antero-posterior section of the excised eye, the tubercular appearance of the iris is most striking. It fills the whole anterior chamber and moulds itself to the shape of the cavity, becoming adherent to the cornea and lens. It presents to the naked eye the appearance of small white nodules massed together and interrupted here and there by the disturbed uveal pigment, giving the growth the resemblance of marble. Microscopically, these nodules present the characteristic features of miliary tubercle, central giant-cells surrounded by epithelioid cells and small round cells. They are non-vascular, though the vessels between them are enlarged and numerous. The ciliary body is usually involved, and the uveal pigment is so interrupted that it would be difficult to believe from the appearance that it was ever a continuous tract. The tubercle bacillus has in many instances been found. Recovery has occasionally taken place with gradual absorption of the growth, but as a rule the prognosis is bad, the eye being lost, and the patient usually dying from general tuberculosis or meningitis. The solitary variety is very similar in its symptoms, pathology, and prognosis, but starts singly.

(2) *Traumatic iritis* results from a perforating injury, mechanical

violence, or chemical irritation. Of these the first is much the most common. The iritis may go on to a suppurative form, with the formation of hypopyon. Suppurative iritis is seldom confined to the tissues of the iris, but is usually either derived from, or extends to, the surrounding tissues.

Under this heading may be included sympathetic iritis; this will be described under sympathetic ophthalmitis.

The treatment of suppurative traumatic iritis is similar to that of suppurative choroiditis.

(3) *Idiopathic iritis.*—Many cases of iritis seem to have no definite cause, either from injury or from constitutional taint. They apparently come on spontaneously, and resemble in every respect an acute plastic iritis or a more chronic irido-cyclitis.

(4) *Secondary iritis* is uncommon. The primary cause is usually a suppurative keratitis; more rarely it follows choroiditis.

Pathology of Iritis and Cyclitis.—In plastic iritis there is hyperæmia of the vessels with small-celled infiltration of the iridic stroma. Exudation follows, consisting of fibrin, with a varying number of leucocytes. This exudation may become completely absorbed, or it may be organised into connective tissue, synechiæ being the result. A similar set of changes takes place in cyclitis, but added to them we have *keratitis punctata.* This condition is found microscopically to be due to small aggregations of round cells cemented together with fibrin, containing a small amount of pigment. They are not formed by any inflammatory condition of the cornea, but result from an inflamed ciliary body, being carried by the lymph stream through the pupil to the back of the lower part of the cornea, where they are usually arranged in the form of a triangle, with the apex at the centre of the pupil.

The complications and sequelæ of iritis are few, though important. *Synechiæ,* anterior and posterior, may occur. The former are almost invariably the result of a perforating wound or of an ulcer of the cornea; rarely they are due to tuberculous or gummatous inflammation of the anterior surface of the iris. The latter may arise from not dilating the pupil early in the attack; exclusion and occlusion are the more serious forms. As a result of annular posterior synechia, atrophy of the iris occurs, with a bulging forwards of the ciliary portion, a condition known as *iris bombé.* This closes the iridic angle, and secondary glaucoma may be set up. Another sequela is opacity of the lens, resulting from deficient nutrition; this may occur with or without *phthisis bulbi,* which is found as a sequela of suppurative iritis following perforation, and in some cases of irido-cyclitis, where there has been much exudation into the posterior chamber and upon the posterior surface of the lens. Lastly, especially in rheumatic iritis, the inflammation may recur, each attack leading to fresh adhesions.

Diagnosis of Iritis.—The more acute form of conjunctivitis may be mistaken for an early attack of iritis. Special attention should be paid to the colour of the iris, its activity, and the condition of the pupil. Glaucoma and iritis have certain signs in common, namely, conjunctival and circumcorneal injection, a muddy appearance of the iris, and a fixed pupil. The history of onset must be carefully investigated; in glaucoma it is much more sudden, and is preceded by characteristic premonitory signs, such as coloured halos seen round artificial lights. The cornea is steamy and anæsthetic in glaucoma; the pupil is dilated rather than contracted; the anterior chamber is shallow, and there is increased intraocular tension.

The diagnosis between iritis alone and iritis accompanied by cyclitis has already been discussed.

The *prognosis* of iritis, if the disease is seen early and judiciously treated, is, in the majority of cases, good, especially if it is syphilitic in origin. Rheumatic iritis is less amenable to treatment, and more liable to recur. If the pupil responds to atropine completely, a good prognosis may be made. On the other hand, the more extensive the synechiæ, especially if there is exclusion or occlusion of the pupil, the graver the condition, secondary glaucoma being very liable to be set up. Occlusion of the pupil results in loss of all useful vision save perception of light. Operative measures in such cases are alone attended with any success; the result may be very decided, but if the condition has existed for years, much improvement cannot be expected. Should the tension be subnormal, operative treatment is contra-indicated. The more chronic the iritis, the more likely are atrophic changes in the eyeball to occur, and so the prognosis is bad. Sympathetic irido-cyclitis and tuberculous iritis are most serious; in the former the restoration of much sight cannot be hoped for, and in the latter enucleation is imperative. Suppurative iritis rarely exists as a local affection, except in septic infection following injury or operation, and consequently the prognosis is unfavourable.

The *treatment* of iritis and cyclitis may be considered under four headings—(1) local; (2) symptomatic; (3) constitutional; and (4) the treatment of complications and sequelæ.

(1) *Local treatment* is of paramount importance, and consists in the dilatation of the pupil, which should be effected immediately the condition is recognised. The best mydriatic is undoubtedly atropine sulphate; it is a vaso-constrictor, thus diminishing the hyperæmia and the exudation; it paralyses the ciliary muscle and the sphincter pupillæ, thus keeping the iris and ciliary body at rest; it prevents the formation of synechiæ and breaks down recent adhesions; and it is an anodyne. At the outset atropine should be used constantly, especially if any synechiæ already exist, when it should be pushed, 0·5, 0·75, or even 1 per cent solutions of the sulphate being successively instilled into the palpebral sac every hour or oftener. This treatment is likely, if anything will, to break down the adhesions, leaving perhaps some pigment upon the lens capsule. Dots of pigment seen in front of the lens are always indicative of previous iritis. If the adhesions have become organised, the atropine will not break them down, but it will prevent the formation of others. When once the pupil has been well dilated, the amount and frequency of atropine administered should be diminished; a solution of 0·25 per cent should now be used three times a day.

The use of atropine occasionally gives rise to atropine irritation, or even atropine poisoning. The former consists of irritable conjunctivitis, and of swelling and erythema of the eyelids and cheek. When this arises the atropine must be stopped at once, and some other mydriatic carefully substituted. For this purpose a $\frac{1}{2}$ per cent solution of sulphate of duboisine, or a 5 per cent solution of hyoscyamine, or a 0·2 per cent solution of scopolamine (formerly known as hyoscine) may be used. Care should also be taken to compress the lachrymal sac with the fingers for a minute or two after the instillation, so as to prevent the fluid from passing down to the nose and pharynx.

Atropine poisoning is recognised by heat and dryness of the mouth and throat, difficulty in swallowing, thirst, and loss of appetite. The face becomes hot and flushed, the pulse is quickened, and the patient may have convulsions, or hallucinations, or be delirious. The pupil of the other eye will be dilated. The atropine must be stopped at once, strong coffee administered, together with a hypodermic injection of a quarter of a grain of morphia or half a grain of pilocarpin.

Besides atropinism there are other contra-indications for the use of atropine and other mydriatics. In cases of irido-cyclitis not only is the tension somewhat raised, but the instillation of atropine is sometimes followed by increased pain, photophobia, and lachrymation. In these cases the atropine solution must be either very weak or replaced by a weak solution of physostigmin.

(2) Of the *symptoms* that must be relieved the chief is pain. Heat, in the form of boracic or belladonna fomentations, is often a great comfort to the patient; dry heat is sometimes more effective than moist. Some patients prefer cold to heat; this may be applied in the form of iced compresses, or by means of a modification of Leiter's tubes. Cocaine may be combined with the atropine; it acts as a sedative, a mydriatic, and a vasoconstrictor. Several leeches applied to the temple close to the outer margin of the orbit often relieve pain and congestion. Each leech will remove rather more than a drachm of blood. Bleeding may be encouraged for an hour after the leeches have dropped off by means of fomentations. Should there be any difficulty in stopping the hæmorrhage, the leech-bite must be touched with lunar caustic and a dossil of lint tightly bandaged over the bites. It is often seen that after the application of leeches the pupil responds better to atropine.

An important aid in the relief of pain, when other methods have failed, is the operation of *paracentesis* of the anterior chamber. It is never attended by bad results, and its value should not be overlooked, especially when the aqueous humour is turbid or contains puro-lymph.

Several measures are important in order that the patient may be placed under the most favourable conditions for a speedy recovery. At the outset a sharp mercurial purge should be given, and throughout the disease a daily action of the bowels must be obtained. If the inflammation is severe, he should be kept in bed in a darkened room, a large double shade or dark goggles being worn. In debilitated subjects tonics will be required, and a light but nutritious diet should be ordered, with alcohol only in strict moderation. In obstinate cases the use of hot air baths causing copious sweating is often very helpful.

(3) *The constitutional treatment* will vary with the etiology.

(*a*) *Syphilitic Iritis.*—It is extremely important that the patient be rapidly got under the influence of mercury, but before this is done certain precautions must be taken. It is important that the mouth should be in a healthy condition. Stumps should be removed, carious teeth filled or extracted, and tartar scraped away. While the drug is being taken the mouth must be kept scrupulously clean. The condition of the kidneys must be ascertained by a careful examination of the urine. During the treatment the patient must be urged to take plenty of good nourishing food, but the amount of alcohol must be small. Chills must be guarded against, and flannel should be worn next the skin.

Mercury may be administered by inunction, inhalation, injection, or by the mouth.

Method of Inunction.—This, perhaps the best method, is performed by rubbing into the skin on the inner side of the arm or thigh, thirty to sixty grains of the blue ointment, the skin being first carefully cleansed. The ointment must be rubbed in for about ten minutes every night just before going to bed, when the patient should be well covered with blankets to produce sweating. Each morning a warm bath may be taken to remove the drug for the day, or a woollen vest may be worn over the same. This treatment should be continued for about six weeks, care being taken to watch against salivation, and after an interval of about three weeks repeated for the same period. During the interval and subsequently mercury should be taken by the mouth.

Method of Inhalation.—Every other night the patient is subjected to the vapour of calomel mixed with steam. Seated on a cane-seated chair, and covered with blankets, he is surrounded by the vaporised calomel, and made to sweat profusely by the accompanying steam. Twenty to sixty grains of calomel should be used each time, and the baths continued till just short of salivation. They should then be stopped and mercury continued to be given by the mouth.

Method of Injection.—The bichloride or biniodide is used for this purpose, not more than a quarter of a grain being injected into the skin or the muscles at each time. Some surgeons largely use and speak highly of intravenous injections followed up by inunctions. Lately Darier has used subconjunctival injections of bichloride of mercury, 1 in 1000, for all deep-seated inflammatory conditions of the eyes, not necessarily syphilitic in origin. Much pain, however, follows in many cases, an adhesive inflammation being set up. It has been found by Mellinger and others that subconjunctival injections of a 2 per cent solution of chloride of sodium have the same beneficial result without any ill effects. Their action depends probably on the acceleration to the lymph current which they produce, and not on any antiseptic or antisyphilitic properties the injection may have. *Vide* article "Gout," vol. iv. p. 237. I have seen several cases of syphilis treated by this recent method, apparently with very satisfactory results. If equally useful, the advantages, from convenience, etc., are very considerable.

Method by the Mouth.—In the treatment of syphilitic iritis this method is usually postponed till the patient has been well brought under the influence of mercury. It may then be continued either in the form of the pill, three grains of blue pill being prescribed, or as a mixture containing the liquor hydrargyri perchloridi with or without iodide of potassium. Treatment should be continued at intervals for eighteen months.

(*b*) *Rheumatic iritis* should be treated with salicin or salicylates combined with alkalies. Iodide of potassium is often useful. Turkish baths may be of great service.

(*c*) The pain in *gouty iritis* is sometimes completely relieved by colchicum; at other times this drug appears to be quite inert. Alkalies should be freely used and the diet carefully attended to.

(*d*) *Tuberculous irido-cyclitis* is rarely amenable to treatment, either local or constitutional; it should be treated by excision of the globe, especially where no other symptoms of tubercle can be discovered in the body.

(*e*) *Gonorrhœal iritis* should be treated with potassium iodide by the mouth, together with local applications to the urethra if any gleet is present.

(4) *Treatment of the Complications and Sequelæ.*—For annular posterior synechia, which leads to secondary glaucoma, an iridectomy must be performed to put the anterior and posterior chambers again into communication. For total posterior synechia a similar operation is usually indicated, but the prognosis is much less favourable; extraction of the lens is often necessary. Chronic recurrent iritis is often cured by an iridectomy.

Isolated posterior synechiæ should be left alone, though formerly they were operated upon by either Passavant's or Streatfield's method (corelysis).

Operations on the Iris.—Iridectomy consists in the excision of a portion of the iris. This operation is performed for various affections of the eye; as a preliminary stage to cataract extraction, for glaucoma, occasionally for purulent infiltration, and in certain forms of ulcer of the cornea, in many cases of chronic and recurrent iritis and irido-cyclitis, and for optical purposes, as in central corneal opacities and stationary lamellar and anterior polar cataracts.

The instruments required are—(1) speculum; (2) fixation forceps; (3) a bent triangular keratome or a Graefe's cataract knife, or a bent broad needle; (4) iris forceps, or a Tyrrel's hook; (5) iris scissors; and (6) a curette.

The operation varies in detail according to the object for which it is performed. The first stage consists in opening the anterior chamber by an incision through the sclerotic close to or at the sclero-corneal junction; the second, in seizing, drawing out, and excising a portion of the iris; the third, in the "toilet" of the wound. When the operation is intended for the relief of glaucoma, or for the purpose of subduing or preventing inflammatory affections, the iridectomy should be made upwards, the coloboma being situated beneath the upper lid. In many cases, such as cataract, chronic glaucoma, and opacities of the cornea, the anæsthetic action of cocaine is sufficient, but where inflammation is actually present, as in iritis, subacute and primary glaucoma, a general anæsthetic is necessary. The patient must lie on a firm table or couch of such a height that his head reaches the level of the umbilical region of the surgeon, who stands behind the patient. The eyelids are kept open by a spring-stop speculum, and the globe is held steady by seizing the conjunctiva and subconjunctival tissue with fixation forceps at the part immediately opposite to that at which the incision is about to be made. A bent triangular keratome is then inserted into the sclerotic at from 1 to 2 mm. from the edge of the cornea, and pushed downwards across the anterior chamber, until the wound thus made is from 6 to 8 mm. wide (Fig. 1). In doing this the instrument is first passed in obliquely, and in such a direction that if continued it would cause a wound of the iris and lens. As soon as its point is seen through the cornea the handle is slightly depressed to bring the blade into a plane anterior and parallel to that of the iris. The keratome is now steadily withdrawn, its apex being kept well away from the plane of the iris and lens.

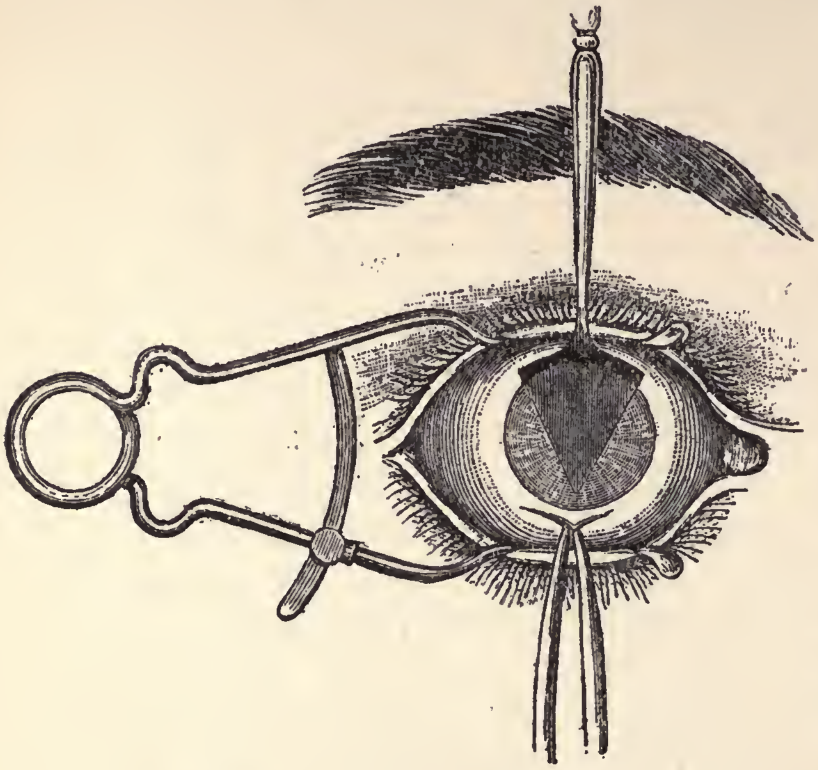

Fig. 1.—The incision with keratome.

The closed iris forceps are now passed into the anterior chamber (Fig. 2), the iris seized near its pupillary edge, dragged just outside the wound, and the projecting portion cut with one snip of the scissors; if necessary, an assistant with fixation forceps slightly rotates the globe downwards without undue pressure or traction. Should, however, a large iridectomy be indicated, the iris is dragged just outside one angle of the wound, and a snip made through its outer part; the portion held in the forceps is then drawn gently across to the other angle, and the excision completed as near to its periphery as possible. Finally, the curette should be passed into the edges of the wound, to liberate any portion of the iris that is entangled there, and the edges of the wound are brought into exact apposition. The speculum is removed, the eyelids gently closed, and a small pad of aseptic gamgee tissue applied by adhesive plaster.

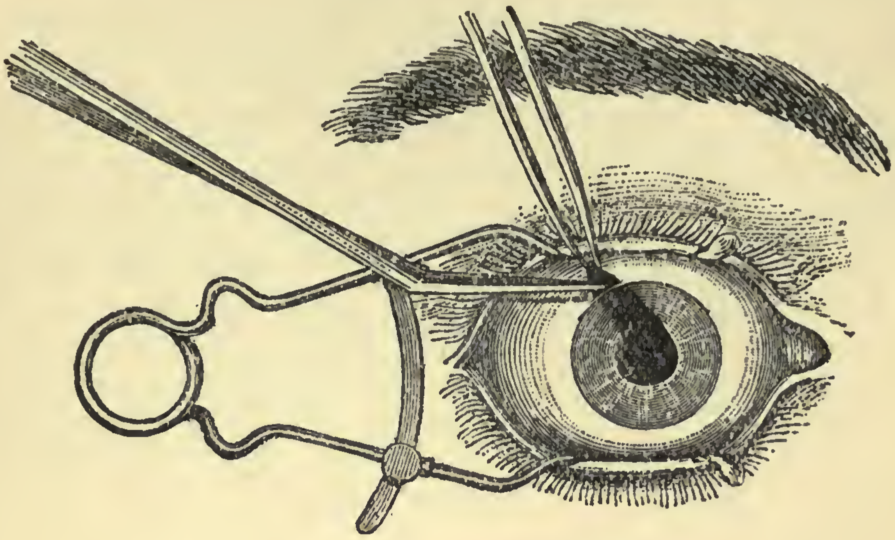

FIG. 2.—The iridectomy.

When the anterior chamber is very shallow the danger of wounding the lens is lessened by the use of a Graefe's knife. Some surgeons always make use of this knife in performing iridectomy.

When the operation is required for optical purposes only, the position of the new pupil necessarily depends upon that of the lesion of the lens or cornea. The best position when possible is downwards and slightly inwards, or straight downwards. Only so much of the iris as is necessary for distinct vision should be removed (Fig. 3). Instead of the large bent keratome a broad bent needle is employed. The position of the incision must depend upon the situation in which the new pupil is required. When this is only slightly excentric it can be made just within the margin of the cornea. When the pupil is required to be opposite the corneal margin the incision must be made in the sclerotic, about 1 mm. beyond the sclero-corneal junction. The width of the incision in either case should be at least 3 mm. Instead of iris forceps a Tyrrel's hook may be used. This is introduced on the flat as far as the centre of the pupil, half rotated downwards, so as to catch the pupillary edge of the iris, then once more turned on the flat, and withdrawn, by which means the iris is extracted.

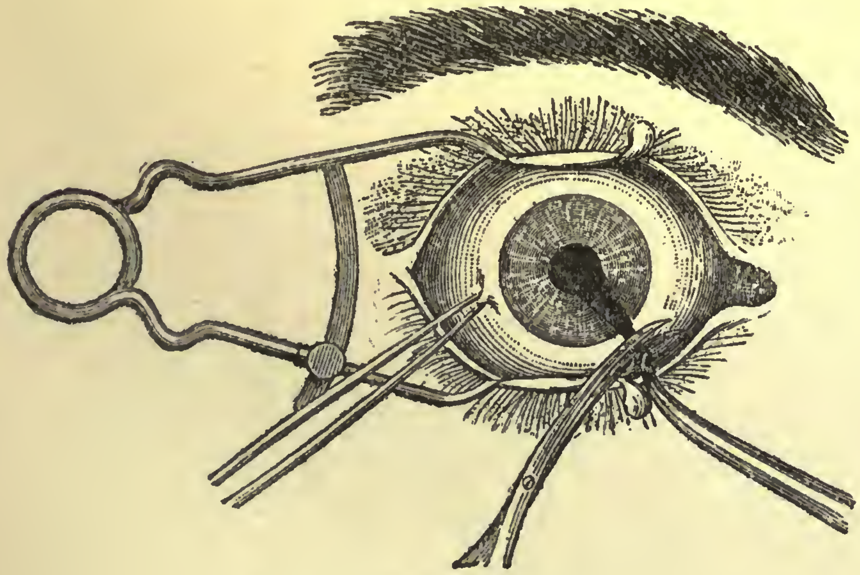

FIG. 3.—Optical iridectomy.

Accidents and Complications of Iridectomy. — (1) The lens may be wounded by the keratome.

(2) The blade of the keratome may get between the corneal layers instead of passing directly into the anterior chamber. This accident arises from it being held too obliquely at the commencement of the incision. As soon as this is discovered the instrument should be immediately withdrawn, and another position selected for a fresh incision. If, however, the blade has finally entered the anterior chamber, so as to cause escape of the aqueous, the eye had better be closed at once, and the operation postponed for twenty-

four hours, that a re-secretion of the aqueous may take place. Without this precaution the iris and lens are so pushed forward after the escape of the aqueous that they are sure to be wounded at the time of the fresh incision.

(3) When the incision is made in the sclerotic there may be considerable hæmorrhage into the anterior chamber either before or after the excision of the iris. The blood can usually be made to flow out by depressing the upper lip of the wound with the curette. When the excision of the iris is completed no anxiety need be felt on account of the presence of a moderate amount of blood in the anterior chamber, as it usually becomes absorbed in a few days.

Iridotomy consists in the formation of an artificial pupil by simple incision of the iris. It can only be safely adopted when the lens is absent, and is mostly applied to those cases in which the iris has become tightly drawn upwards towards the cicatrix, as the result of inflammation after cataract extraction.

The eyelids being separated, and the globe steadied, as in the previous operations, a small keratome is passed through the upper part of the cornea about 2 mm. from the sclero-corneal junction; it is then pushed onwards through the membranous exudation to the back of the iris, and finally withdrawn. The iridotomy scissors with closed blades are now passed through the corneal wound. When they reach the iris one blade is passed behind and the other in front of that structure, which is now divided by a single snip from above downwards. In some cases it is necessary to make a second incision at an acute angle with the first, so as to include a V-shaped piece of iris, which can either be left to atrophy or removed.

TUMOURS OF THE IRIS AND CILIARY BODY.—Primary sarcoma of the iris is very rare. About half of the recorded cases are melanotic. The diagnosis of the leucotic variety from tubercle is impossible; the melanotic form may be mistaken for melanoma, which, however, is stationary and non-vascular. Metastasis is common. The eye should be enucleated as early as possible.

Primary sarcoma of the ciliary body is even more rare.

Primary Carcinoma of the Ciliary Body.—A few cases of this disease have been recorded. The cells are pigmented, and are probably derived from the ciliary glands.

Tubercle and gumma of the iris and ciliary body are described under inflammatory conditions.

Cases of melanoma and nævus of the iris have been recorded.

Cysts of the iris are of three varieties, all of which are rare.

(1) *Epithelial Cysts.*—These are probably always implantation cysts, in some case a hair, in others corneal epithelium, being carried by a perforating injury into the iridic stroma. They are lined by squamous epithelium and contain epithelial débris. They appear some months or even years after the injury, grow slowly, press on the cornea, producing opacity, and encroach on the pupil, causing loss of sight. Ultimately they may set up glaucoma. When small they should be removed by an iridectomy. If not seen till late, enucleation or evisceration may be necessary.

(2) *Serous cysts* differ pathologically from epithelial cysts, though their mode of origin is not definitely known. Some suppose that they are formed from the crypts on the anterior surface of the iris, the outlets being occluded by chronic inflammatory exudation; others consider their formation to be secondary to the formation of synechiæ. Microscopically they are lined by a single layer of epithelium. Their course, symptoms, and treatment are similar to those of epithelial cysts.

(3) Cysts have been described as occurring between the two layers of the uvea. Iritis usually supervenes.

INJURIES TO THE IRIS AND CILIARY BODY.[1]—Severe contusion of the eyeball without perforation may injure the iris in several ways. *Rupture of a blood-vessel* may be the result of the blow. This is characterised by the presence of blood in the anterior chamber (hyphæma). The eye must be carefully examined for other lesions of the iris and of other structures, but it may be necessary to wait till the blood has become absorbed. Should other lesions be absent the prognosis is good, and the blood will be absorbed in a few days. The eye should be kept at rest by atropine and a shade, and no reading should be attempted. Very rarely iritis supervenes.

Traumatic mydriasis is probably always due to some laceration of the iris, and is consequently usually accompanied by hyphæma. The laceration is radial, and may only implicate the pupillary border, being often so minute as to require high magnification before it can be seen. Paralysis of the sphincter pupillæ, *iridoplegia,* results. Combined with it may be paralysis of accommodation, *cycloplegia.* Iridoplegia may occur without cycloplegia, but traumatic ciliary paralysis alone is rare. The treatment consists in rest in bed and the application of cold. Atropine should not be used unless iritis, which is very uncommon, is threatened. Later, pilocarpine is indicated. The prognosis must be guarded, as the condition is often permanent.

Irido-dialysis, or detachment of the iris from the ciliary body, is usually only partial. The ciliary portion of the iris is its weakest part, and consequently is subjected to the greatest strain when the cornea is flattened by a blow. Hyphæma is always present, and, if severe, may hide the detachment. The circulus arteriosus iridis major is probably ruptured, together with Schlemm's canal. The appearance is that of two irregular pupils. Ophthalmoscopically the ciliary processes and the suspensory ligament of the lens, unless this has been ruptured, may be seen. The prognosis is not good, as the condition is usually permanent, though few cases of spontaneous cure have been reported. The treatment should be iced compresses and atropine, which will help to approximate the edges of the gap.

Traumatic aniridia or irideremia is the name given to total irido-dialysis. The totally detached iris floats forwards and sinks to the lower part of the anterior chamber.

Retroflexion and *anteflexion* are very rare dislocations of the iris, and are usually only partial. In retroflexion a part of the iris is doubled back so as to cover the ciliary processes, the suspensory ligament being ruptured with dislocation of the lens. The appearance differs from that of a coloboma, since the ciliary processes cannot be seen. In anteflexion the ciliary border of the iris is not only detached from the ciliary body, as in irido-dialysis, but is doubled forwards over the rest of the iris, the posterior pigmented layer coming into view.

Penetrating wounds of the anterior part of the eyeball, unless quite small, are usually followed by prolapse of the iris, or of the ciliary body, or of both.

Traumatic Prolapse of the Iris.—This is especially found in wounds of the sclero-corneal junction. Besides the prolapse of the iris the lens may be injured. Strict antiseptic precautions should be taken in the treatment. The patient should be put to bed, and the wound carefully cleansed with perchloride of mercury lotion, 1 in 5000. The protruding portion of the iris must be excised, and the edges of the wound freed as much as possible from the iris to prevent the formation of anterior synechia. Atropine must be instilled into the eye three times a day. These cases often do well, very useful vision being regained. Should pyogenic organisms enter the eye, however, at the time of the injury or subsequently, suppurative iritis is very liable to be set up, followed by panophthalmitis.

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[1] See also "Eyeball, Injuries of," vol. iii.

Traumatic prolapse of the ciliary body is of much more serious import. If only slight it may be possible to suture the sclerotic or conjunctiva over it, but on no account should it be removed, as vitreous will then escape. If at all extensive, with collapse of the eyeball, there is very great danger of sympathetic ophthalmitis being set up. Consequently the eye should be removed without delay.

Perforation of the eyeball with a *foreign body* in the iris or ciliary body is very serious, since it is imperative that the foreign body should be removed as soon as possible. If this is impossible the eye must be enucleated or eviscerated without delay. Of the two a foreign body in the iris is much the more favourable. The diagnosis is seldom difficult. It is best removed through an incision at or near the periphery of the cornea. If magnetic, the nozzle of an electro-magnet should be introduced through the incision.[1] If not magnetic, or if the electro-magnet fails to remove it, it is well to proceed very cautiously in attempting its removal by means of small forceps. In attempting to remove a small particle of non-magnetic substance from the iris it is safer to remove a portion of the iris as well as the foreign body. The small iris forceps should be introduced through an incision of the cornea made in the vicinity of the particle as far away from the centre of the cornea as possible. Each blade of the forceps should then be made to grip the iris on either side of the particle, so as to include the latter in iris tissue, the forceps are then withdrawn through the incision, and the iris cut off with iris scissors as in ordinary iridectomy, when with good luck the particle will be found inside the excised portion of iris. The temptation is to try and pick the foreign body off the iris, but this usually fails, and causes immediate hæmorrhage into the anterior chambers, so that the particle is obscured, and is probably detached from its first position.

A foreign body in the ciliary body is often difficult to diagnose, and always extremely dangerous. Occasionally the history is sufficiently definite to make the diagnosis certain, but where this is not the case recourse must be had to other methods of investigation. During the last few years the discovery of the Röntgen rays has caused great strides to be made not only in the diagnosis of, but also in the exact localisation of foreign bodies in the eye and orbit. When thus localised the body must be cut down upon through the sclerotic with strict antiseptic measures, extracted, and the incision sutured. Another method, applicable, however, only to magnetic substances, is by the use of the large electro-magnet of Haab, which will often bring foreign bodies from the ciliary body into the anterior chamber, or even completely out of the eyeball. But in spite of these recent additions to the diagnosis and treatment, this condition remains very serious, and often necessitates evisceration or enucleation.

Other results of a penetrating injury are *implantation* and *serous cysts*, which are referred to previously (p. 518).

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[1] See also "Eyeball, Injuries of," vol. iii.

Itch. *See* Scabies.

Jacksonian Epilepsy. *See* Epilepsy.

Jaundice.

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Definition.—Jaundice is the condition due to the presence of bile pigment in the blood, and is recognised clinically by staining of the skin, conjunctivæ, mucous membranes, and urine by bile pigment.

Causation.—The formation of bile pigment from hæmoglobin is carried on by the cells of the liver, for, as shown by experiments on animals, this transformation does not take place when the liver is removed or, what comes to the same thing, when all the vessels going to it are ligatured. Jaundice, or the presence of bile pigment in the blood, is due to the bile manufactured by the liver passing into the circulation instead of into the intestines. This miscarriage of the bile may occur either directly the bile pigment is formed by the liver cells, *i.e.* before the bile enters the bile ducts, or later after it has entered the ducts.

In cases of long-standing biliary obstruction, where the dilated intrahepatic ducts only contain mucus, the bile formed by the liver cells passes almost directly into the adjacent lymphatics, thence into the thoracic duct, and so reaches the general circulation. Cases of this kind, in which no bile is visible in the ducts, were formerly thought to prove that jaundice might result from suppression of the bile-excreting function of the liver, and be due to the resulting accumulation in the blood of bile pigment formed in the circulation from hæmoglobin. The presence of bile pigment in the liver cells in such cases shows that the formation of bile still continues.

When there is obstruction in the ducts the bile passes from the bile capillaries or ducts into the lymphatic vessels of the liver, and not into the blood-vessels. This has been shown by the experiments of Saunders early in this century (1809), and later by Fleischl (1874), V. Harley (1892), and Szubinski (1899). It has also been shown that jaundice due to ligature of the bile duct may be removed or prevented by ligature of the thoracic duct. Obstruction to the flow of bile through the ducts leads to a rise of the normally low pressure of bile, and as a result the bile passes into the lymphatics, and so into the general circulation.

This clearly explains the production of jaundice in cases where a gross mechanical obstruction in the ducts exists, but it is necessary to consider the method of causation of jaundice in those cases where there is no manifest obstruction in the larger bile ducts.

Pathology of Jaundice

Jaundice was formerly divided into (1) hepatogenous or obstructive, where there was an unmistakable obstruction in the larger ducts, and

(2) Where there was no gross obstruction visible to the naked eye. In contrast to the first category, this form was spoken of as non-obstructive. It included cases—(*a*) Where the jaundice was thought to be due to changes occurring in the blood, leading to the liberation of hæmoglobin and its transformation in the circulation into bile pigment. This constituted "hæmatogenous" jaundice. (*b*) Where an excessive secretion of bile into the intestine was followed by excessive absorption from the mucous membrane of the bowel and subsequent overflow into the general circulation. This was called jaundice from polycholia. (*c*) Where owing to suppression of the bile-excreting function of the liver cells an accumulation of bile pigments, manufactured from hæmoglobin in the blood, occurred in the circulation. This was known as jaundice by suppression.

Consideration of "Hæmatogenous" Jaundice.—In various toxic and septic conditions of the blood, where the hæmoglobin is liberated from the red blood corpuscles, jaundice of a slight degree of intensity is often seen. A good example of this "toxæmic" jaundice is provided in the experiments of Hunter with toluylenediamine. This poison, when introduced into the circulation, gives rise to destruction of the red blood corpuscles with liberation of hæmoglobin (hæmolysis), and to jaundice. After its administration the flow of bile is at first increased, and there is an increase in the bile pigments poured out (polychromia), owing to an augmented amount of free hæmoglobin, the antecedent of bile pigments, reaching the liver. After a time the amount secreted diminishes, and the bile becomes more viscid, until finally the flow of bile becomes almost arrested. This slowing and diminution in the flow of bile is found to depend on inflammation of the smaller intrahepatic bile ducts, which become swollen, secrete thick mucus, and are in a condition of catarrhal cholangitis due to the toxic effects of the toluylenediamine. The jaundice, therefore, is really obstructive, but being due to changes in the small intrahepatic ducts is readily overlooked. This explanation accounts for the slight jaundice accompanying morbid conditions such as septicæmia, pyæmia, hæmoglobinuric fever, pernicious anæmia, and poisoning by phosphorus, arseniuretted hydrogen, and snake-bite. This jaundice, therefore, is toxæmic, and depends on the presence of a poison in the blood which sets up inflammatory changes, and consequently obstruction in the small intrahepatic ducts. It may therefore be conveniently described as hæmohepatogenous, but not as hæmatogenous jaundice. Experiment shows that the presence of free hæmoglobin in the blood, though it leads to an increased secretion of bile pigment in the bile (polychromia), does not of itself give rise to jaundice. The frequent clinical association of jaundice with conditions giving rise to destruction of red blood corpuscles is due to the fact that the hæmolysis is of toxic origin; inflammation of the small intrahepatic bile ducts is another manifestation of the toxæmia.

The Question of so-called Jaundice from Polycholia.—When jaundice was found to be associated with bile in the fæces it was supposed that there was such an excessive secretion of bile (polycholia), that an excessive amount of bile was absorbed from the intestines and passed through the liver into the general circulation.

Bile may be present in the fæces of jaundiced patients under various conditions; thus, in obstruction of one hepatic duct icterus results, but the other hepatic duct pours bile into the duodenum. When a calculus lies in a common duct it may act like a ball valve, and allow bile to escape into the bowel at intervals; in biliary cirrhosis bile is usually present in the fæces. In none of these examples is there any suggestion to the effect that an excessive secretion of bile exists.

In hæmohepatogenous or toxæmic jaundice there is in the early stage a secretion of bile rich in bile pigment (polychromia) and poor in bile acids, the pigment appearing in the excreta; later obstruction in some of the small intrahepatic ducts occurs and produces jaundice. This is the explanation of so-called jaundice from polycholia. But it should be pointed out that, strictly speaking, there is not an excessive secretion of normal bile (polycholia), but only of bile pigment (polychromia), and that the bile salts, far from being increased, are diminished.

The Question of the so-called Jaundice from Suppression of the Bile-secreting Function of the Liver.—It was formerly supposed that the liver might, from nervous or other influences, cease to secrete bile, and that an accumulation of refuse blood-pigment in the circulation resulted, and became changed, without the intervention of the liver, into bile pigment. Emotional jaundice was instanced, but there is no proof of the liver thus striking work, and moreover, inasmuch as bile is only formed by the liver, if its bile-forming functions were suppressed jaundice could not result. This last argument, indeed, is fatal to the idea of jaundice from suppression of the bile-forming function of the liver. In acute yellow atrophy and phosphorus poisoning the jaundice is almost certainly due to obstruction in the small intrahepatic ducts from inflammation, and to the passage of bile into the lymphatics, which results as a natural consequence. In long-continued biliary obstruction the ducts contain clear mucous fluid devoid of bile, and it might, and indeed has been thought, that the liver ceased to secrete bile under these conditions; but here the liver cells, as shown by the presence of bile pigment inside them, still manufacture bile, which passes almost directly into the lymphatic vessels and so into the circulation.

"Urobilin Jaundice."—A few words may be added on this subject. Darkening of the skin may occur in the melanæmia of malaria, in hæmochromatosis, and in other conditions, but does not really resemble jaundice. It has, however, been thought that a staining of the skin indistinguishable from jaundice may be due to other pigments than those of bile. Thus in some cases where there is an excessive amount of urobilin in the urine a yellow tinging of the skin has been referred to the effects of urobilin, and not of bile, in the circulation. Excessive urobilinuria may occur without any jaundice; when they are associated together the jaundice is due to bile pigment and not to staining of the skin with urobilin. As Hunter points out, the term urobilin *jaundice* is a misnomer, and moreover is confusing and misleading.

Classification.—Jaundice may be divided, therefore, into—

(1) Obstructive—due to some gross obstruction to the flow of bile along the larger bile ducts.

(2) Toxæmic or hæmohepatogenous jaundice, where there is obstruction in the small intrahepatic ducts, due to poisons excreted from the blood into the bile ducts setting up cholangitis.

Jaundice is therefore always due to obstruction, and is a symptom, not a disease in itself. It may be a symptom of a purely local condition, viz. pressure on the large ducts, or, on the other hand, be the result of a general infection or intoxication setting up changes in the small intrahepatic bile ducts resulting in obstruction.

TOXÆMIC OR HÆMOHEPATOGENOUS JAUNDICE

General Characters and Distinctions from Obstructive Jaundice.—Toxæmic or hæmohepatogenous jaundice is essentially a symptom of some underlying infection, or possibly intoxication, and is usually subordinate to its other constitutional symptoms. The patient suffers comparatively little from the direct results of bile pigment circulating in the blood, but is definitely ill from the general toxæmic state.

The jaundice is slight as a rule; in acute yellow atrophy and icterus gravis the yellow colour may be bright and marked, but the dark green or black tint of chronic obstructive jaundice does not occur.

Bile pigment is found in the urine. It has usually been stated that bile acids are either absent, or at any rate not present to the same extent as in jaundice due to mechanical obstruction of the larger ducts; but little importance can be attached to results obtained by Pettenkoffer's well-known test, since, as shown by Hunter, it is entirely unreliable as employed in ordinary clinical practice.

The motions nearly always contain bile, and thus differ from the clay-coloured stools of icterus due to obstruction.

The course of the disease is more acute as a rule than in obstructive icterus, and is not accompanied by the itching of the skin, xanthopsia or yellow vision, and slow pulse that may occur in obstructive jaundice.

Signs of constitutional disturbance, such as enlarged spleen, fever, and albuminuria, are common, while grave symptoms develop sooner and more frequently than in obstructive jaundice, where when they do occur it is at the termination of long-continued jaundice.

In severe cases of hæmohepatogenous jaundice the "typhoid state," with dry tongue, delirium, coma, and multiple hæmorrhages, may rapidly develop.

The important features, therefore, are the slight degree of jaundice and the presence of marked constitutional symptoms, the want of proportion between the icterus and the symptoms, and the evidence of hæmic infection or intoxication.

After Hunter, hæmohepatogenous or toxæmic jaundice may be divided into—

(i.) Toxic jaundice, due to poisons, such as toluylenediamine, arseniuretted hydrogen, phosphorus, snake poison.

(ii.) That occurring in connection with the specific fevers, such as yellow fever, pyæmia, septicæmia, relapsing fever, and malaria.

(iii.) A group containing acute yellow atrophy, icterus gravis, infectious jaundice (Weil's disease).

Obstructive Jaundice

Signs.—Jaundice appears first in the conjunctiva, then successively on the face, body, and extremities. The "whites" of the eyes are the first part to show the bile pigment, but before this bile pigment is present in the urine. The masses of fat (pinguecula) often seen under the conjunctivæ frequently have a slightly yellow colour, and may mislead a careless observer into the belief that jaundice is present. A slight degree of icterus is more readily detected in a fair-skinned patient than in one of a dark complexion, in whom the skin is often somewhat sallow. It should also be remembered that jaundice is easily overlooked in artificial light. The mucous membrane of the lips and palate shows an icteric tint. When jaundice has existed for a long time the bile pigment in the skin turns of a dark green colour (biliverdin), and the skin becomes of an olive-green colour, spoken of as "black jaundice." After recovery from severe jaundice the skin is often left discoloured for a very considerable period.

In jaundice continued for more than a year the skin may show the peculiar change known as xanthoma or xanthelasma.

When as the result of complete biliary obstruction, or from other causes, the protective function of the liver fails, and toxins pass into the circulation and give rise to cholæmia or biliary toxæmia, hæmorrhages may take place into the skin and mucous membranes, giving rise to petechiæ, epistaxis, melæna, etc.

The urine is acid in reaction, somewhat diminished in amount, and becomes bile-stained before the conjunctiva or skin. There may be also a considerable excess of urobilin. Stress was formerly laid on the presence of bile acids in the urine as a point of importance in the diagnosis from jaundice occurring without manifest obstruction to the ducts. The test relied on for the detection of bile acids in ordinary clinical work, Pettenkoffer's, is so fallacious that no value can be attached to its result, and hence the question of bile acids in the urine is no longer of any practical importance. The colour of the urine varies in different cases and at different times, from an intensified yellow to brown, olive, or a very dark brown. When shaken up the froth becomes yellow. The colour must be distinguished from that in urobilinuria, hæmaturia, melanuria, and the alteration effected by rhubarb and chrysophanic acid, by senna and santonin, by employing Gmelin's test for bile pigment. When cholæmia or biliary toxæmia has supervened, the renal epithelium may be so damaged by toxins that albuminuria results.

The other secretions as a rule do not contain bile; sometimes the sweat, especially in the axilla, is undoubtedly bile-stained, but generally the perspiration is colourless. The secretion of the intestines and nose, the saliva, tears, and in women, milk, are, in spite of statements to the contrary, free from bile. In inflammatory conditions the pathological secretions and exudations become icteric, as shown by pneumonic sputum, pleural and peritoneal effusions, and saliva in mercurial salivation.

In obstructive jaundice there is constipation, and the motions are extremely offensive. They are clay-coloured from absence of bile and the presence of an excess of fat, the digestion of which is interfered with.

Exceptionally bile may be present in the fæces, for example, when jaundice is due to obstruction to one of the two hepatic ducts, where a calculus in the common duct allows some of the bile to escape into the duodenum.

The Circulatory System.—In the absence of fever or pain the pulse tends to be slow; this has been ascribed to the action of bile salts on the cardiac ganglia. It is, however, especially in catarrhal jaundice that the slowing is chiefly marked.

The blood-pressure is low. Though from the destructive (hæmolytic) action of bile acids on red blood corpuscles anæmia might be expected, it is not a marked result of jaundice itself. From muscular incompetence a mitral systolic murmur may become audible, and from increased pressure in the pulmonary circulation due to this cause, or possibly to reflex constriction of the pulmonary vessels referred from the bile ducts, the second sound over the pulmonary artery becomes accentuated.

The liver is often enlarged from damming up of the bile, and may be tender. In malignant disease and cirrhosis its surface may be knobby or irregular.

Enlargement of the gall-bladder is a valuable sign of obstruction of the common duct. When chronic jaundice is due to gall-stones, the gall-bladder is collapsed from previous cholecystitis, while in cases of malignant disease pressing on the common bile duct the gall-bladder may form a tense, pear-shaped tumour. If a calculus be impacted in the cystic duct the gall-bladder may be distended with mucus, and occasionally it is enlarged from the presence of numerous calculi; these are exceptions to the general rule enunciated by Courvoisier, that in jaundice due to gall-stones the gall-bladder is collapsed.

The spleen is usually not enlarged, but if it is it points to biliary cirrhosis, some septic or toxic process, such as Weil's disease or infective jaundice, to syphilis, or to that extremely rare condition, alveolar hydatid.

The temperature, like the functions of the body generally in simple obstructive jaundice, is depressed. Fever, when present, is either due to the same cause that is responsible for the jaundice, as in toxæmic jaundice, Weil's disease, and cirrhosis, or is the result of some complication, such as cholangitis in gall-stone obstruction.

Symptoms.—The symptoms that may be met with accompanying jaundice are due partly to the presence of bile in the circulation, which acts as a depressing poison, partly to a secondary toxæmia due to the liver failing to perform its important protective function of stopping poison brought to it from the alimentary canal, and partly to the absence of bile from the alimentary canal. These distinctions in the causation of the symptoms must not be pressed too far, but it will be convenient to consider the symptoms under the following heads:—

Symptoms due to Bile in the Circulation.—Bilirubin is said to be ten times more toxic than the bile acids. It will be noticed that in cases of jaundice a considerable amount of the bile pigment is fixed in the tissues, which become thereby bile-stained; in this way the rest of the body is protected from the full toxic effects of all the bile pigment that has entered into the circulation. The tissues in which the bile pigment settles suffer; for example, in the skin the irritation of the cutaneous nerves shows itself by pruritus, which may be very troublesome and prevent sleep. Scratching thus induced may be the cause of pimples and traumatic eczema, while urticaria and lichen are sometimes seen.

A bitter taste in the mouth is often complained of, although the saliva does not contain bile pigment; this bitter flavour may depend on the presence of toxic bodies, which, owing to hepatic insufficiency, have escaped into the general circulation and passed into the saliva, and if so, would properly come in the next category.

There is often considerable impairment of appetite with a special distaste for fatty foods.

Mental depression, low spirits, and incapacity for continued thought or application, are commonly seen.

Yellow vision or xanthopsia due to the media of the eyes being jaundiced, is sometimes found to be present on inquiry, but is hardly ever a prominent symptom. It is indeed better marked after the ingestion of santonin than in jaundice.

Symptoms due to the Presence of Poisons other than Bile in the Circulation.—Owing to the failure of the liver to stop poisons received from the alimentary canal, auto-intoxication results, and if the kidneys do not compensate for this by free diuresis, a toxæmic condition, analogous to that of uræmia, results. Drowsiness, delirium, and somnolence passing into coma may develop.

Symptoms due to the Absence of Bile from the Intestinal Tract.—Constipation is very common, and is usually explained as due to the absence of the natural purgative—the bile. Excessive fermentation with flatulence and extremely offensive motions may occur, while interference with the digestion and with the absorption of fatty food leads to an excessive amount of fat in the fæces (steatorrhœa).

Diagnosis.—As has already been pointed out, jaundice may be overlooked altogether if the patient is only seen by yellow artificial light, such as gas or candle illumination. No serious difficulty should arise in distinguishing jaundice from other pigmentary changes in the skin.

The yellow colour of patients with slight icterus is hardly likely to be confused with the bronzing of sunburn, with the natural hue of the yellow-skinned races of mankind, or with the tint of the skin in the advanced cachexia of malignant disease, but should any question arise, examination of the conjunctivæ, and of the urine for the presence of bile pigment, will quickly settle the matter.

The dark green colour of the skin in black jaundice of long-continued biliary obstruction might conceivably be confused with the pigmentation of the skin in Addison's disease, hæmochromatosis, malarial melanæmia, argyria, etc., but here again examination of the urine and conjunctivæ will prevent error.

The past effects of severe jaundice may give rise to some difficulty, but further investigation and the history of the case should make matters clear.

Differential Diagnosis of Jaundice due to various Causes.—Since the distinction between obstructive jaundice and toxamic (or hæmo-hepatogenous) jaundice has already been considered (p. 523), it is now only necessary to refer to the differential diagnosis of the various factors that may give rise to obstructive jaundice.

The causes of obstructive jaundice are very numerous, but for convenience they may be grouped into three classes:—

(1) Where the obstruction is due to something occluding the lumen of the bile ducts, such as a gall-stone, or parasites.

(2) Where the obstruction depends on changes originating in the walls of the larger bile ducts, *e.g.* catarrhal cholangitis.

(3) Where obstruction is due to processes arising outside the ducts and occluding them, either (i.) by direct pressure alone, or (ii.) by actually invading the tissues of the ducts.

(1) *Causes obstructing the Lumen of the Ducts.*—(*a*) A gall-stone in the common duct is the most familiar example. A history of biliary colic, often of repeated attacks, preceding the development of jaundice is the rule; sometimes, however, no history of colic is forthcoming, and the case may at first very closely resemble malignant disease, but as time goes on the jaundice is not uniformly or necessarily progressive; sometimes, as the duct dilates around the stone, the jaundice fades and may even disappear. Another point of distinction is that in cholelithiasis the gall-bladder is usually small and contracted from past inflammation of its walls, whereas in malignant disease it is often enlarged. It is sometimes found that gall-stones and malignant disease are combined, especially in the case of the gall-bladder.

When gall-stones remain long in the common duct infective cholangitis with intermittent hepatic fever may develop (*vide* "Disease of Gall-bladder and Bile Ducts," vol. iv. p. 46).

Inspissated mucus and small calculi may also give rise to slight jaundice and attacks of pain, often described as "spasms," and regarded as dyspepsia.

(*b*) *Parasites.*—A hydatid cyst may discharge into the ducts, and membrane or daughter cysts block the ducts, giving rise to biliary colic, jaundice, and often to infective cholangitis. The diagnosis depends on the history that the patient has either had a cyst which has disappeared, or that an existing one has grown smaller about the time that the symptoms appeared, or still better on the presence of bile-stained pieces of hydatid membrane in the fæces, or possibly even in the vomit.

Round worms (ascaris lumbricoides) may enter the common bile duct from the duodenum and give rise to jaundice. Of this rare condition Mertens has collected forty-eight examples.

Distomum hepaticum, the liver fluke so fatal to sheep, has been found in the ducts of the human liver, as have in rare instances D. sinense and D. conjunctivum. In these cases the diagnosis depends on the recognition of the worms or their ova in the fæces.

(*c*) As pathological curiosities some authors have referred to the presence of foreign bodies, such as needles, in the bile ducts.

(2) *Changes originating in the Walls of the Ducts.*—(*a*) Catarrhal jaundice due to inflammatory swelling of the mucous membrane of the duct, at or extending some distance from the biliary papilla, and due to the spread of inflammation from the duodenum, is preceded by gastro-intestinal disturbance—diarrhœa and vomiting. It is of short duration, one to two weeks, and is not accompanied by the fever, enlarged spleen, and albuminuria seen in infectious jaundice. It should, however, be borne in mind that the jaundice of malignant disease and of acute yellow atrophy may begin exactly like benign catarrhal jaundice.

(*b*) The infective and suppurative forms of cholangitis are usually associated with gall-stones or the rupture of a hydatid cyst into the ducts; but the more severe form complicates a considerable proportion of the cases of that rare condition, primary carcinoma of the duodenal surface of the biliary papilla.

Possibly jaundice in the roseolous stage of syphilis is due to a specific cholangitis.

In the infective forms the symptoms are those of intermittent hepatic fever, while in suppurative cholangitis the clinical picture is that of suppuration in the liver, and closely resembles pylephlebitis. It may be incidentally pointed out that jaundice may be absent in the suppurative cholangitis.

(*c*) Growths of the mucous membrane of the ducts.

Carcinoma, which is sometimes met with, gives rise to deep progressive jaundice with enlargement of the gall-bladder. At first, since it is often accompanied by colic, it may resemble gall-stones, while later it resembles malignant disease of the head of the pancreas.

Innocent tumours, such as papillomata, are curiosities and cannot be accurately diagnosed.

Xanthelasma has been known to occur in the mucous membrane of the bile ducts, but if multiple its occurrence on the skin would probably, and in most instances rightly, be referred to jaundice due to some other cause.

(*d*) Simple stricture due to the cicatrisation of past ulceration in the ducts, such as might conceivably be set up by the passage of a gall-stone, is so extraordinarily rarely shown to exist that, clinically, cases of jaundice following hepatic colic should be regarded as due to fresh gall-stone impaction or to malignant disease.

In very rare instances cicatrisation of a duodenal ulcer involving the biliary papilla has been demonstrated, but duodenal ulcers are usually confined to the first part of the duodenum, and are often latent. Jaundice following the symptoms of duodenal ulcer would therefore more reasonably be regarded as due to adhesions involving the common duct.

Congenital obliteration of the bile ducts, giving rise to persistent icterus in young children, has already been described (*vide* vol. iv. p. 57). The age of the patient and its persistent nature make its diagnosis easy.

(*e*) Spasm of the muscular fibres of the ducts explains emotional jaundice, such as that due to fright, satisfactorily, but inasmuch as it is difficult to prove, its occurrence is open to question.

(3) The conditions that may press on or invade the bile ducts from without, and so interfere with the flow of bile and give rise to jaundice, are very numerous. A tumour may merely press on the duct, or on the other hand it may actually invade the walls of the duct; a floating kidney may exert temporary traction or pressure on the ducts, or inflammatory adhesions spreading out from a gastric, duodenal ulcer, or other focus of inflammation, may involve the ducts and strangle them. The following list shows the great variety of conditions that may be met with:—

(*a*) Tumours projecting from the liver itself into the portal fissure may compress the bile ducts. Thus a carcinomatous mass may compress the ducts; exceptionally it occludes one hepatic duct and sets up jaundice, though bile from the other lobe enters the duodenum and colours the fæces. A hydatid cyst may, though it rarely does, press on the ducts. A gumma occasionally arises in this situation and sets up jaundice.

(*b*) Enlarged glands in the portal fissure may press on the ducts. The enlargement may be secondary to carcinoma of the stomach, colon, gall-bladder, liver itself, pancreas, kidney, etc. Enlargement may also be due to tubercle or possibly to syphilis.

(*c*) Malignant disease may spread by continuity up the lesser omentum from the pylorus and give rise to jaundice by invading the ducts. Jaundice is said to be met with in 5 per cent of cases of gastric carcinoma, and may depend either on a direct extension of the growth or on a secondary growth in the portal fissure.

(*d*) As already mentioned, fibrosis spreading out from a gastric ulcer at the pylorus, or even from a duodenal ulcer, may involve and compress the ducts. It is rare, and when it does occur is likely to be regarded as malignant disease, since the matting together of the tissues may be palpable as a tumour. It is remarkable that jaundice is hardly ever seen in perihepatitis or in simple chronic peritonitis—conditions in which fibrosis and cicatricial contraction are very marked, and might be expected to compress the bile duct.

(*e*) Enlarged glands in the neighbourhood of the common bile duct near its entrance into the duodenum may be secondary to carcinoma in various organs. When the duct is thus obstructed the gall-bladder should be enlarged, and the condition closely resembles malignant disease of the head of the pancreas.

(*f*) *Tumours and Cysts of the Pancreas.*—Malignant disease of the head of the pancreas, which is practically always spheroidal-celled carcinoma of comparatively slow growth, forms a hard mass, rarely large enough to be felt, which compresses and may invade the common bile duct. The duct becomes dilated, and the gall-bladder often forms a tense, pear-like tumour. The jaundice is progressive and deep, and emaciation is rapid.

Hydatid cysts have been known to occur in the head of the pancreas, and to give rise to jaundice.

Pancreatic and peripancreatic cysts rarely come so far to the right as to compress the ducts, but exceptionally this does occur. Cysts of the pancreas are readily felt, and should be distinguished from a distended gall-bladder by the fact that the stomach, if inflated, is found to lie in front of the cyst.

(*g*) Retroperitoneal tumours arising from glands, connective tissues, and possibly from accessory adrenal bodies, may grow forward and involve the bile ducts. As in the case of pancreatic and renal tumours, coils of intestine may intervene between them and the abdominal wall, and give rise to a resonant note on percussion.

(*h*) *Renal Tumours.*—A primary tumour of the right kidney rarely presses on the bile duct, giving rise to jaundice; a secondary growth in the portal fissure is a more probable cause of jaundice in primary renal growths. The same is true of primary tumours in the suprarenal bodies.

Floating kidney on the right side may give rise to attacks of biliary colic and jaundice; the condition is therefore very likely to be regarded as cholelithiasis. The mechanism has been thought to be due to direct pressure of the kidney on the duct, but Macalister regards it as due to traction, the peritoneum covering the kidney being continuous with that around the bile duct.

(*i*) *Abdominal Aneurysms.*—An aneurysm of the aorta under the pancreas may compress the common bile duct and give rise to jaundice.

Aneurysms of the hepatic artery are rare, but when they occur are frequently accompanied by jaundice. The aneurysm may compress the common hepatic or hepatic ducts, in which case the gall-bladder will not be dilated as it is when an aortic aneurysm compresses the common duct.

Aneurysm of the commencement of the superior mesenteric artery has been known to give rise to jaundice.

Aneurysms can only be diagnosed as the cause of jaundice when they can be felt.

(*j*) *Glenard's Disease.*—In rare instances hepatoptosis may give rise to twisting of the bile duct and jaundice. Dilatation and ptosis of the stomach may also induce kinking of the bile duct, or by leading to kinking of the duodenum prevent the outflow of bile from the biliary papilla.

(*k*) *Uterine and Ovarian Tumours.*—Large fibro-myomata and cysts have been known to produce jaundice, but they must reach an enormous size before doing so, and their existence would be manifest long before.

Malignant tumours arising in the uterus or ovaries may, by means of secondary growths in or near the liver, give rise to biliary obstruction.

(*l*) Fæcal impaction in the colon has been said to give rise to jaundice, but the association is rare, and it is probable that when these two conditions are associated there is either some underlying cause, such as malignant disease, which accounts for them both, or the jaundice is due to catarrh of the ducts.

Differential Diagnosis

The large number of conditions of which jaundice may be a symptom makes it essential that a careful examination should be made for evidence of disease elsewhere. Thus the existence of a tumour in the abdomen, breast, or rectum will suggest malignant disease, while the coexistence of syphilis, in either the secondary or tertiary stage, should be an indication for specific treatment.

The following points have a bearing on the nature of jaundice in a given patient:—

Age.—Transient slight jaundice noticed a few days after birth is physiological, but if the jaundice is well marked, and accompanied by fever, septic infection spreading from the umbilicus may have taken place. Persistent jaundice from birth is in favour of congenital obliteration of the ducts.

In early adult life catarrhal jaundice should be thought of. Between the ages of 30 and 40 years gall-stones, especially in women, are the most probable cause, while later in life malignant disease and cirrhosis must also be taken into account.

Sex.—Women are more prone to gall-stones and to malignant disease, men to cirrhosis, and perhaps to the infectious forms of jaundice, such as Weil's disease, which have been noticed to attack soldiers and butchers.

Pregnant women seem more susceptible than others to acute yellow atrophy.

Onset.—If preceded by gastro-intestinal disturbance, catarrhal jaundice is indicated; if by severe colic, gall-stones. A gradual onset with no special accompaniments should suggest the pressure of a tumour, practically always of a malignant nature, on the ducts. Repeated and transient attacks are in favour of gall-stones.

Occurrence of Pain.—Constant pain is suggestive of malignant disease, intermittent attacks point to gall-stones. Attacks of biliary colic may also occur when hydatid membranes are passed through the ducts, and occasionally when malignant disease involves the ducts—pseudo-gall-stone colic.

Absence of pain is, however, no proof against malignant disease, though it is the rule in catarrhal jaundice, and common in cirrhosis.

Duration and Progress.—Jaundice of short duration is most commonly catarrhal, or due to the passage of gall-stones. If continued for more than six months it is highly unlikely that malignant disease is present, and biliary cirrhosis or impacted gall-stone should be thought of. Jaundice lasting for years is probably due to biliary cirrhosis.

Progressive and black jaundice suggests malignant disease, while jaundice that persists but varies from time to time is more compatible with a stone impacted in the common duct, or with biliary cirrhosis.

Degree and Intensity of Jaundice.—Slight icterus may be catarrhal or, if associated with fever and constitutional conditions, toxæmic. The commonest causes of deep jaundice are malignant disease, impaction of a gall-stone in the common duct, and biliary cirrhosis. Extremely deep jaundice almost necessitates malignant disease; that accompanying gall-stone impaction is deeper than that of cirrhosis, but never equals that seen in compression of the common bile duct by tumours, such as carcinoma of the head of the pancreas, or in malignant disease of the duct.

Outbreaks of jaundice in epidemics suggest some form of toxæmic jaundice.

Fever suggests toxæmic jaundice, or biliary cirrhosis when associated with considerable splenic enlargement; pyrexia, of course, occurs in calculous cholangitis, in hepatic suppuration such as abscess or pylephlebitis, and occasionally in malignant disease.

Considerable enlargement of the liver is met with in biliary cirrhosis, in abscess, and in malignant disease; in the latter it is progressive, and the surface will probably be nodular.

The association of ascites with jaundice points to malignant disease or to cirrhosis, but in the former jaundice is deeper. In fact the darker the jaundice the more likely is the cause to be malignant growth.

The existence of disease elsewhere in the body has already been insisted on as of supreme importance in arriving at a diagnosis of the cause, and therefore of the prognosis and treatment of jaundice.

PROGNOSIS.—The occurrence of jaundice in certain diseases is of importance as showing that hepatic complications have supervened; thus when, as very rarely happens, jaundice is met with in the course of typhoid fever, cholangitis or cholelithiasis should be thought of. In puerperal eclampsia, jaundice is of extremely bad omen; death follows in a few hours or days. The onset of jaundice after phosphorus poisoning is a sign that the liver is affected, and must be regarded as of the most grave significance, since few cases recover when this stage is reached. In like manner the occurrence of jaundice in malaria is of very serious import, as pointing to a severe hæmic infection, such as occurs in hæmoglobinuria or "blackwater" fever.

The ultimate prognosis of any case of jaundice is of course dependent on the cause, and not on the degree of the jaundice; thus the slight icteric tint seen in toxæmic jaundice due to pyæmia, and the black jaundice of complete obstruction due to malignant disease, do not differ very much with regard to their prospect. The prognosis of chronic jaundice is bad; it is worst in cases of complete obstruction by malignant disease, for in addition to the fatal nature of the growth, biliary toxæmia or cholæmia is likely to occur; it is rather better in hypertrophic biliary cirrhosis, for although complete recovery is very improbable life may yet be prolonged for a very considerable time. In chronic jaundice due to gall-stones the prognosis is fair because operative measures can be undertaken with a fair prospect of success.

When chronic jaundice is accompanied by xanthelasma or by the bulbous or "Hippocratic fingers" it may be assumed that the cause of the jaundice is not malignant disease, inasmuch as this would have killed the patient before these changes could have had sufficient time to develop.

In chronic jaundice much depends on the functional activity of the kidneys being well maintained; if the amount of urine falls and waste products are less freely excreted biliary toxæmia is likely to result. The presence of albuminuria points to the kidneys being damaged by the toxæmia accompanying the jaundice, and is therefore an index of a severe condition. The detection of leucin and tyrosin in the urine of a case of jaundice makes the prognosis very grave.

When jaundice is accompanied by hepatic insufficiency, so that poisons which should have been destroyed by the liver escape into the general circulation, and give rise to a general toxæmia as shown by nervous symptoms such as delirium, drowsiness, and coma, and by hæmorrhages, the prognosis is very grave indeed, since life cannot be long maintained after the development of cholæmia. The occurrence, therefore, of nervous symptoms in cases of jaundice should always arouse anxiety.

There are cases at first quite indistinguishable from simple catarrhal jaundice in which nervous symptoms somewhat rapidly develop, and the case then runs the course of acute yellow atrophy of the liver. Again, in other instances the jaundice associated with malignant disease of the liver may begin exactly like catarrhal jaundice. Caution is therefore necessary in forming a prognosis in the early stages of what appears to be simple catarrhal jaundice, especially in patients past middle life.

Treatment.—The radical and most satisfactory plan is, of course, the removal or cure of the cause of the jaundice; for this an accurate diagnosis of each case is essential. The results of operations for the removal of gall-stones show how successful this plan may be.

Even in cases of inoperative malignant disease, such as carcinoma of the head of the pancreas, surgical measures may still give some relief by removing the extreme jaundice. Cholecystenterostomy, or uniting the gall-bladder to the small intestine, has been performed, and by allowing the bile to enter the intestine has removed the jaundice, prevented the occurrence of biliary toxæmia or cholæmia, and greatly improved the patient's condition for a time. The operation must be performed early; if black jaundice has already developed, the patient is in a very unfavourable state for this procedure.

The Palliative Treatment of Jaundice.—Symptoms should be treated as they arise; constipation should be met by five grains of blue pill or calomel, followed by a saline; want of appetite by dilute acids and nux vomica; nausea and vomiting, if present, by bismuth, dilute hydrocyanic acid, and morphia. If there is much flatulence, creasote, salicylate of bismuth, liquor perchloridi hydrargyri, and salol may be employed as intestinal antiseptics.

Capsules of ox bile, or pills coated with keratin so as to prevent the bile being liberated in the stomach, may be given before food three times daily in order to replace the absent bile in the intestine.

For the pruritus alkaline baths or sponging with carbolic lotion 1 in 40 should be tried. Pilocarpine $\frac{1}{6}$-$\frac{1}{4}$ of a grain injected hypodermically often gives good results. For itching of the skin and hæmorrhages the administration of chloride of calcium in 15 or 20 grain doses may be tried three times a day for two or three days, but not longer at a stretch, as its effect in promoting coagulation of the blood is lost after a comparatively short time.

Before an operation such as a cholecystotomy on a jaundiced patient, it is well to give chloride of calcium so as to diminish the risk of bleeding from the jaundiced tissues.

The patient should be encouraged to take plenty of water so as to avoid constipation, and in catarrh or cholangitis to wash out the ducts; in these cases salicylate of soda may also be taken with advantage inasmuch as it increases the secretion of bile and so tends to sluice out the bile ducts.

A milk diet should be adhered to since it is easily digested, tends to minimise intestinal fermentation and auto-intoxication, and acts on the kidneys as a diuretic. Fatty food for which the patient often has a distaste should be avoided.

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JAUNDICE OF PHOSPHORUS POISONING

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Usually phosphorus poisoning is suicidal, and is effected by consuming rat pastes or making a meal of the heads of lucifer matches. It is rare in England; in the ten years ending 1892 there were 147 fatal cases in England and Wales (W. Blythe), but it is not infrequent abroad, especially in Vienna. It may be due to rat paste accidentally swallowed, or, as in Leonard Hill's case, be due to the application of phosphorescent paste to the skin.

It sometimes follows the medicinal use of the drug; the smallest fatal dose for an adult is $\frac{1}{8}$ of a grain; usually 1 to 2 grains, either taken in a single or in divided doses, is the quantity followed by a lethal result. In a baby the smallest fatal dose known is $\frac{1}{100}$ grain.

Results very similar to those of phosphorus poisoning may follow poisoning with iodoform, arsenic, antimony.

Morbid Anatomy.—The liver is, as a rule, much larger than natural, firm but friable, and of a pale yellow colour. In a few cases the liver has presented exactly the features of acute yellow atrophy, but this is exceptional, and the change in the liver is essentially one of increase in size due to acute fatty change, and resembles that of iodoform poisoning. It is said that as time goes on the liver diminishes in size.

Under the capsule and on section the yellow buff aspect of the liver substance shows here and there reddish spots due to hæmorrhage, which stand up against the bile-stained liver substance.

In dogs poisoned by phosphorus there is a large quantity of fat in the liver, and the nuclei of the liver-cells show fragmentation. There is, however, no fat or almost none in the myocardium, none in the voluntary muscles, and only a little in the kidneys (Ray, M'Dermott, and Lusk).

Microscopically the liver-cells show cloudy swelling and very advanced fatty change. The cells in parts of the lobules may contain granules of bile pigment. Leucin and tyrosin may be found in the liver. There is sometimes slight proliferation of the connective tissue elements of the portal spaces, and in cases that recover some cirrhosis may develop.

The heart and kidneys show advanced fatty change; the heart may be so soft as to be readily perforated by the fingers during its examination. The voluntary muscles also undergo fatty change.

The spleen may be much enlarged.

Hæmorrhages are found scattered throughout the body.

The *symptoms* due to the irritating effect of phosphorus on the gastric mucous membrane come on from a few minutes to three hours after taking the poison. With phosphorated oil or phosphorus in a soluble state the ill effects are soon manifested, while if the poison was taken in a solid form the onset is delayed. There is gastric pain and vomiting, which may be so constant as to lead to dangerous collapse. The vomited matters and eructations may be luminous in the dark. The vomit may become dark and grumous. There is usually intense thirst, tenderness over the stomach and liver, but no hepatic enlargement at this stage.

The patient may die from collapse; if he does not, and has been satisfactorily treated, permanent recovery may follow, but in a considerable number of cases there is a temporary improvement succeeded by the onset of grave symptoms due to the toxic effects of the absorbed poison on the liver and other internal organs.

These severe symptoms usually begin about four days after the poison was taken; they may come on sooner, or, on the other hand, be delayed for two, three, or even six weeks, as in S. West's case.

It begins with jaundice and recurrence of vomiting of dark, grumous matters, then follows great prostration, ending in coma and death, usually on the fifth or sixth day from the time the phosphorus was taken, and after a day or two of grave constitutional symptoms.

Hæmorrhages into the skin and from mucous membranes are a constant feature of the disease, but are not so marked as in deep jaundice due to other causes. A blood count shows an increase in the number of red blood corpuscles in the circulation.

Jaundice is by no means a constant symptom, and does not, when present, bear any relation to the severity of the changes taking place in the liver. From experiments on animals, and from some observations in man, there is reason to believe that, as in acute yellow atrophy, the jaundice is obstructive and due to inflammation and stagnation of viscid bile in the smaller ducts.

The temperature is usually below normal.

The liver is enlarged and tender, the spleen is also enlarged, and the abdomen may become distended.

The most marked difference between this stage of phosphorus poisoning and acute yellow atrophy is in the size of the liver. But in exceptional cases of phosphorus poisoning the liver is not enlarged, and in the early stages of some examples of acute yellow atrophy it is enlarged, so that the clinical resemblance between the two affections may sometimes be very close.

The urine is somewhat diminished in quantity, but never suppressed; it is high-coloured and of rather high specific gravity. It may contain albumin, blood, and casts. There are generally bile pigments and bile acids, while sarcolactic acid is very frequently present. It was formerly said that leucin and tyrosin were not present in the urine, and stress was laid on this in the diagnosis from acute yellow atrophy; it is now known that leucin and tyrosin may be present in the urine in phosphorus poisoning, but are far from being constantly found. Leucin is less frequently found than tyrosin. The amount of urea was formerly thought to be greatly diminished; it now appears that though it is diminished in the early stages when all food is refused by the stomach, it is absolutely increased in the later stages as a result of increased metabolism. Experimentally it has been shown that the rise in the proteid metabolism in phosphorus poisoning is only equalled by that in phloridzin diabetes. It is said that alimentary glycosuria is more readily induced in phosphorus poisoning than in health, but it is very seldom found in practice; Hunter says that only three cases are on record.

Diagnosis.—The history that phosphorus has been swallowed, or that symptoms justifying this conclusion have recently occurred, is a most important, if not essential point. The presence of phosphorus in the vomit, or the fact that the vomited matters are luminous in the dark, of course settles the question.

In the differential diagnosis from acute yellow atrophy the following points should be borne in mind:—(i.) The large size of the liver. (ii.) The amount of urea is not diminished except in the earliest stage. (iii.) The greater prominence of gastro-intestinal symptoms.

Prognosis.—In cases where jaundice and enlargement of the liver come on, the outlook is very grave; most cases die.

Treatment.—When the poison has been recently taken the stomach should be emptied and washed out.

As an antidote French or old or oxidised oil of turpentine should be given every quarter of an hour for the first hour, 40 minims in an emulsion, and afterwards three or four times daily.

Permanganate of potash and sanitas have also been recommended. Mucilaginous drinks should be given. But oils or fats should be avoided, as they render the phosphorus more soluble. Purgatives should be given.

When the grave constitutional symptoms have developed no special treatment can be relied on.

LITERATURE.—Blythe, W. *Poisons*, p. 213; 1895.—Hunter. Allbutt's *System*, vol. iv.—Mann, J. Dixon. *Forensic Medicine and Toxicology*, 2nd ed. p. 499.—Ray, M'Dermott, and Lusk. *American Journal of Physiology*, vol. iii. p. 139, Oct. 1899.—West. *Lancet*, 1893, vol. ii.

Jaws.—When we consider the important rôle played by the upper and lower jaws in health, the importance of their rôle in disease is obvious. According to popular belief the set of the lower jaw, by which is meant the state of development and tenacity of its bony and muscular structures, is an important index of character—that is, an index of the state of mental development of the individual. In this connection it is interesting to refer to the views entertained by some alienists, that a similar imperfect state of development of the upper jaw, as revealed by a characteristic arch of the palate, is not infrequent in many cases of mental deficiency, more especially in those which first manifest symptoms about the end of the first decade. This view is by no means fully confirmed by the experience of dental practitioners, and the subject will be again referred to. (See "Teeth.")

The diseases of the lower jaw are almost entirely surgical, including various forms of inflammation, tumour growths, and cystic formations, involving the mucous membrane of the gums, the periosteum, or the bone. The main points of more medical interest are those relating to affections of the temporo-maxillary joint (see "Rheumatism" and "Rheumatoid Arthritis"), the different forms of neuralgia, and the later stages of paralysis when the "jaw jerk" is a marked clinical symptom. (See "Neuralgia" and "Paralysis.")

Diseases of the upper jaw have special significance because of the various irregular cavities that lie amongst the bones of the face and the intercommunications between these different cavities.

When we bear in mind that the upper jaw is the principal bone of the face, entering into the formation of the floor of the orbit, the floor and lateral wall of the nasal cavity and the hard palate, in addition to constituting the site for the attachment of the upper teeth, with their important functions and delicate nervous supply, we need not wonder that the exact diagnosis of the position and nature of the different diseases encountered is sometimes very difficult to determine. This is specially true of the various suppurative diseases of the antrum of Highmore and the various accessory cavities communicating with it. This will be discussed in the section on "Nose," where the differential diagnosis of disease in the different accessory cavities will be fully considered. The other general surgical articles will be mainly considered in the article on "Mouth," to which the reader is referred.

END OF VOL. V

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